

# Gastroduodenal Surgical Diseases and Gastric Cancers



Dr. Osama H. Alsallaq  
MD. General and Oncology Surgeon MRCS  
European Board of Surgical Oncology  
Department of Surgery  
King Hussein Cancer Center (prev.)  
Mu'tah University

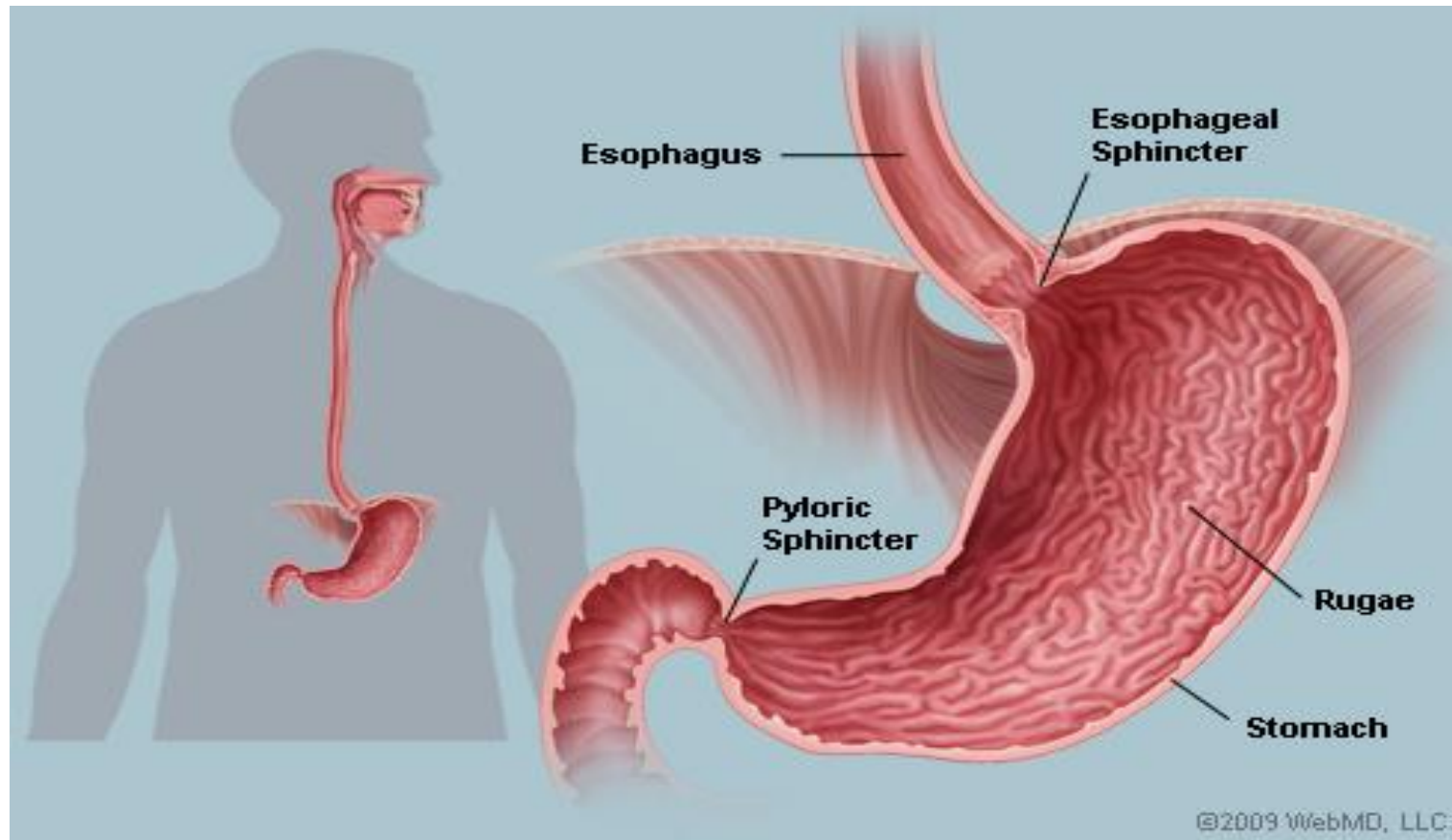


# Outlines

- ▶ Surgical anatomy of the stomach.
- ▶ H.PYLORI infection
- ▶ Peptic ulcer disease
- ▶ Post gastrectomy syndromes
- ▶ Gastric adenocarcinoma
- ▶ Gastric lymphoma
- ▶ Gastrointestinal stromal tumors (GIST)



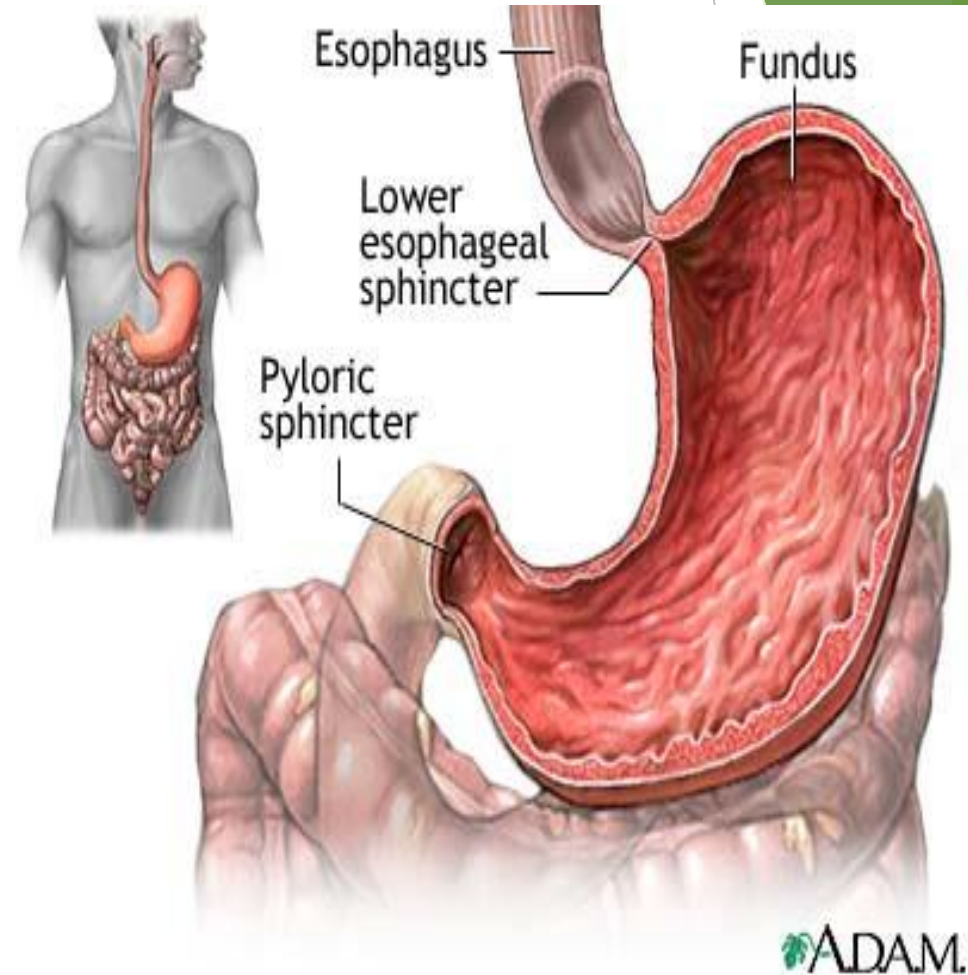
# STOMACH ANATOMY





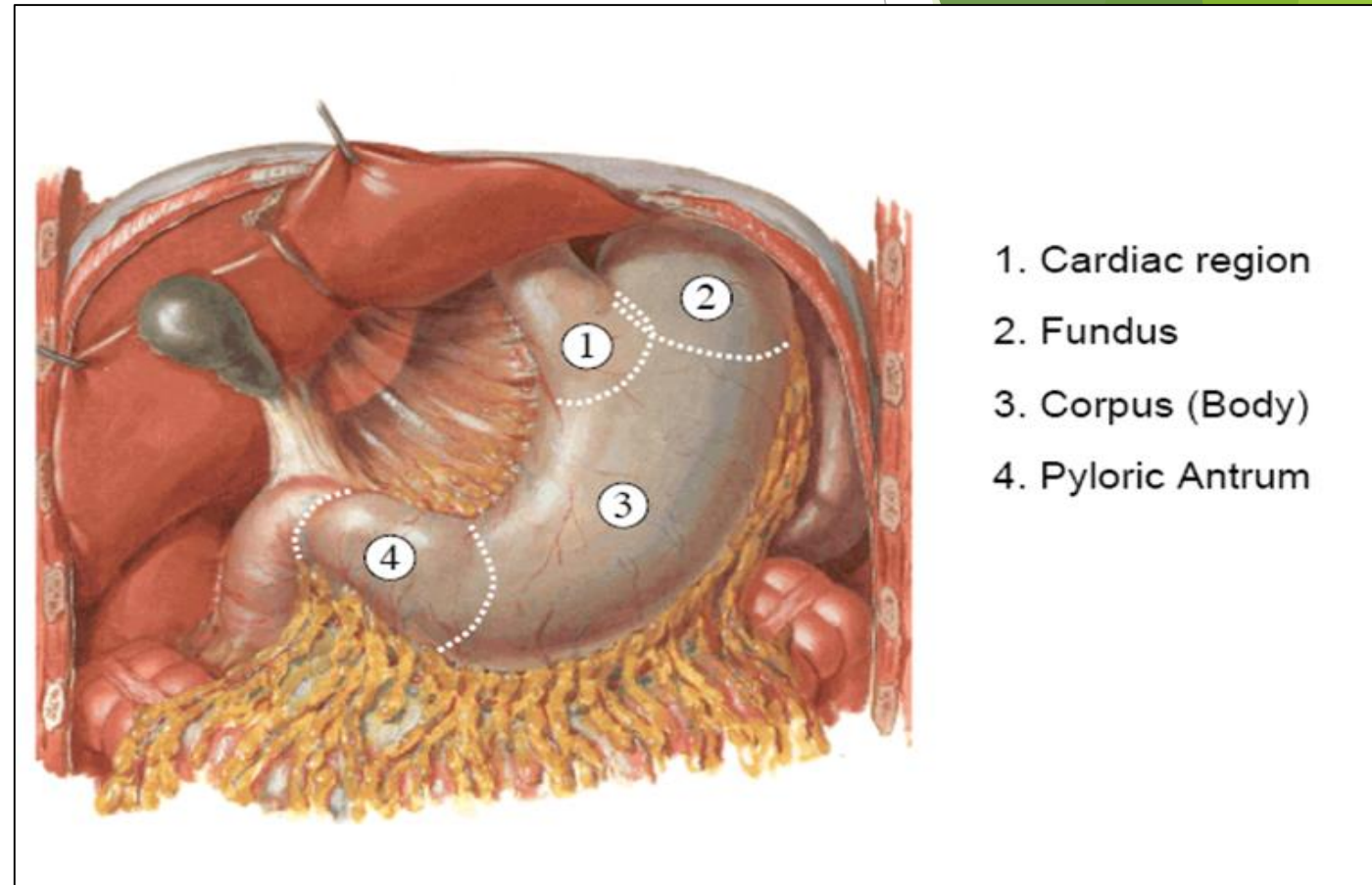
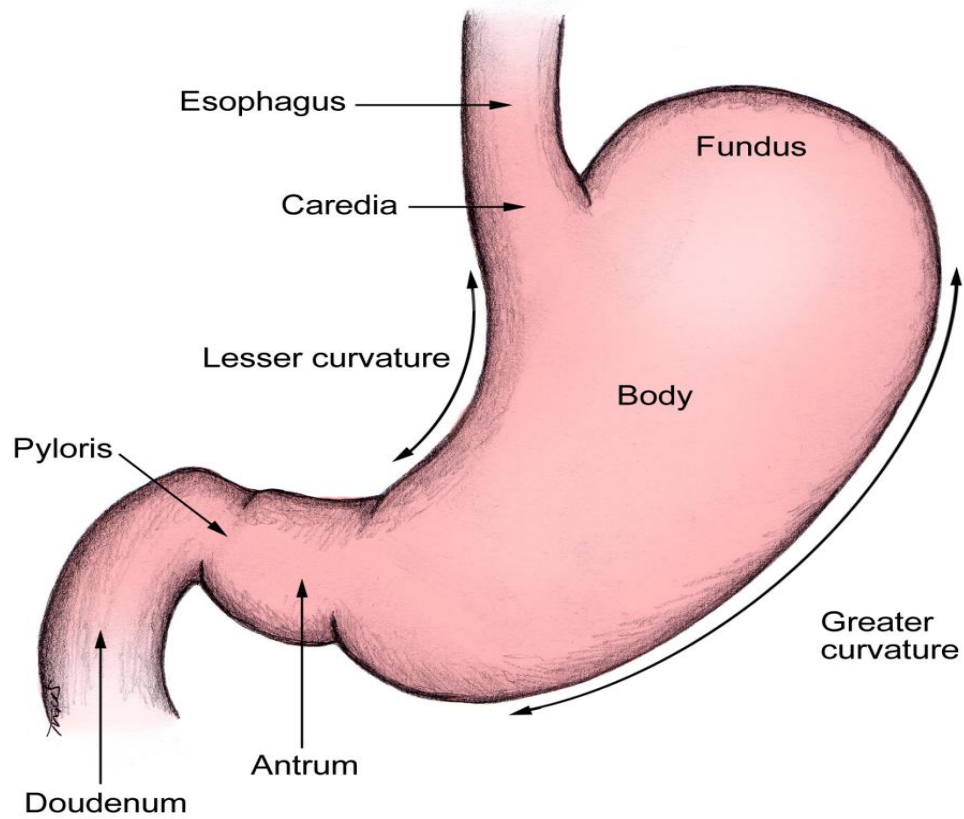
# The stomach

- ▶ The stomach is a dilated part of the alimentary canal between the esophagus and the small intestine.
- ▶ It is a J-shaped muscular sac.
- ▶ located at level of T10 and L3 vertebral.
- ▶ Position of the stomach varies with body habitus





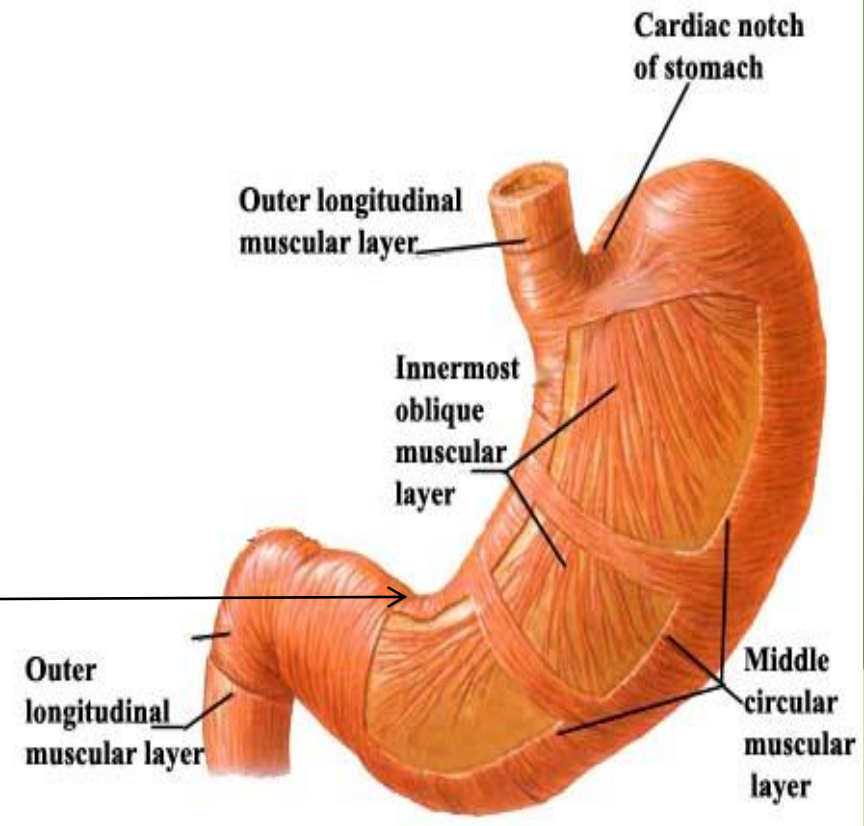
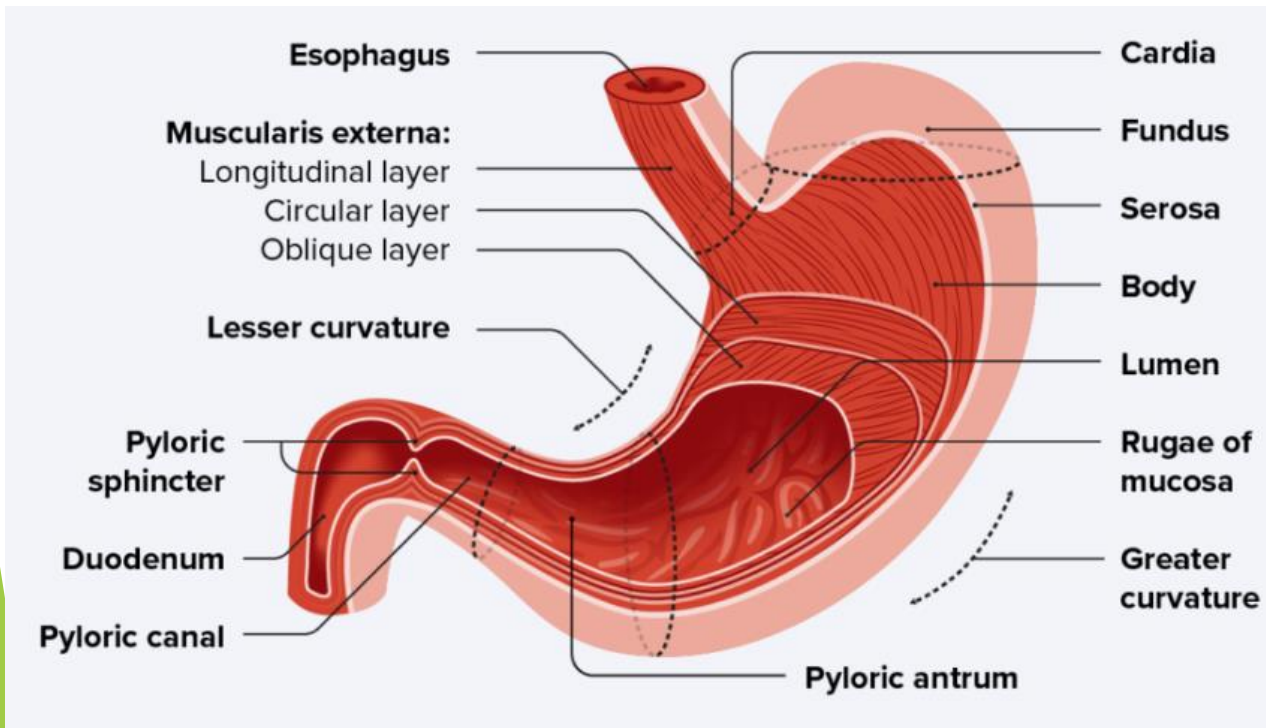
# The Stomach





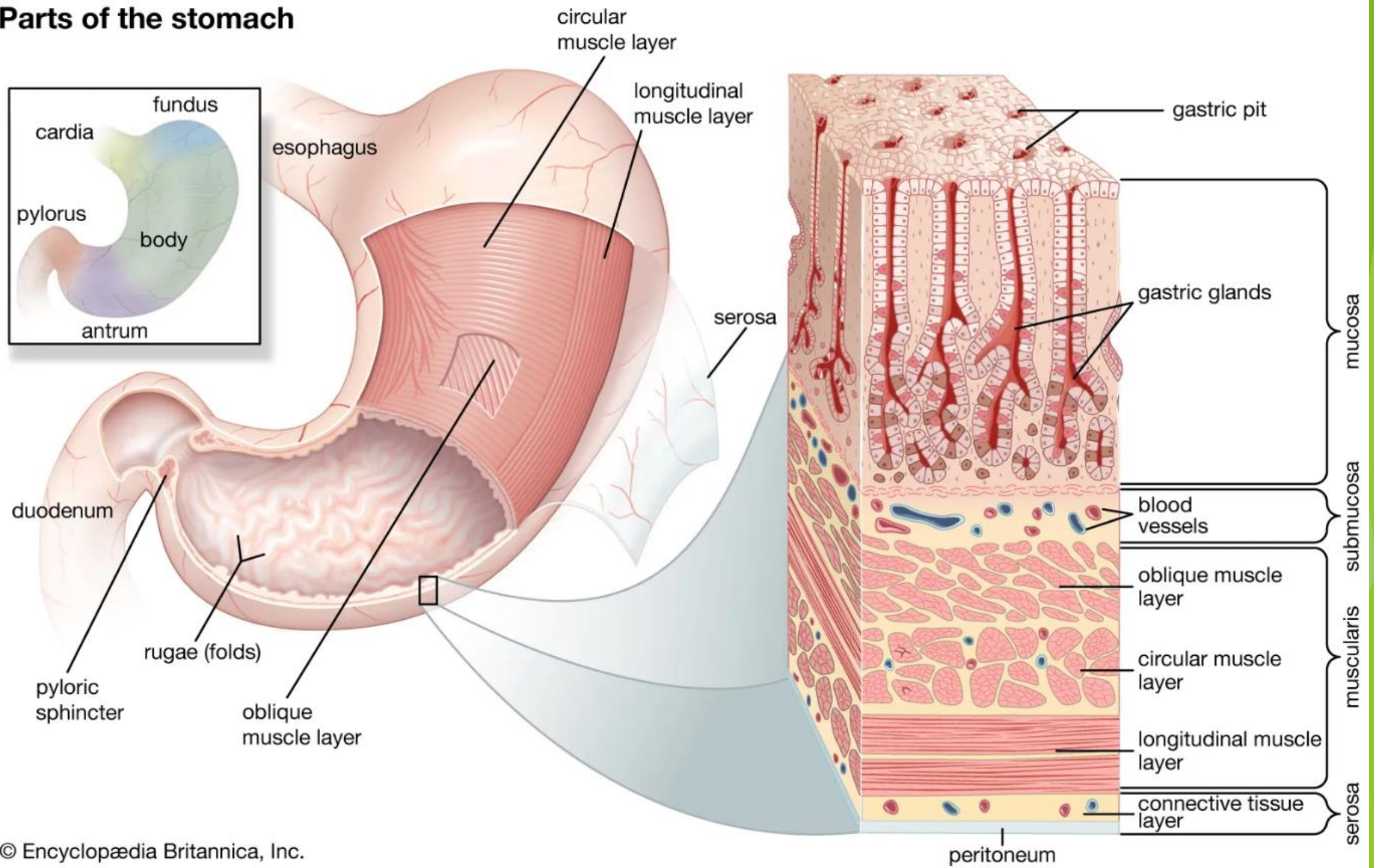
# The stomach

- ▶ the **cardial notch**, which is the superior angle created when the esophagus enters the stomach.
- ▶ the **angular notch**, which is a bend on the lesser curvature.





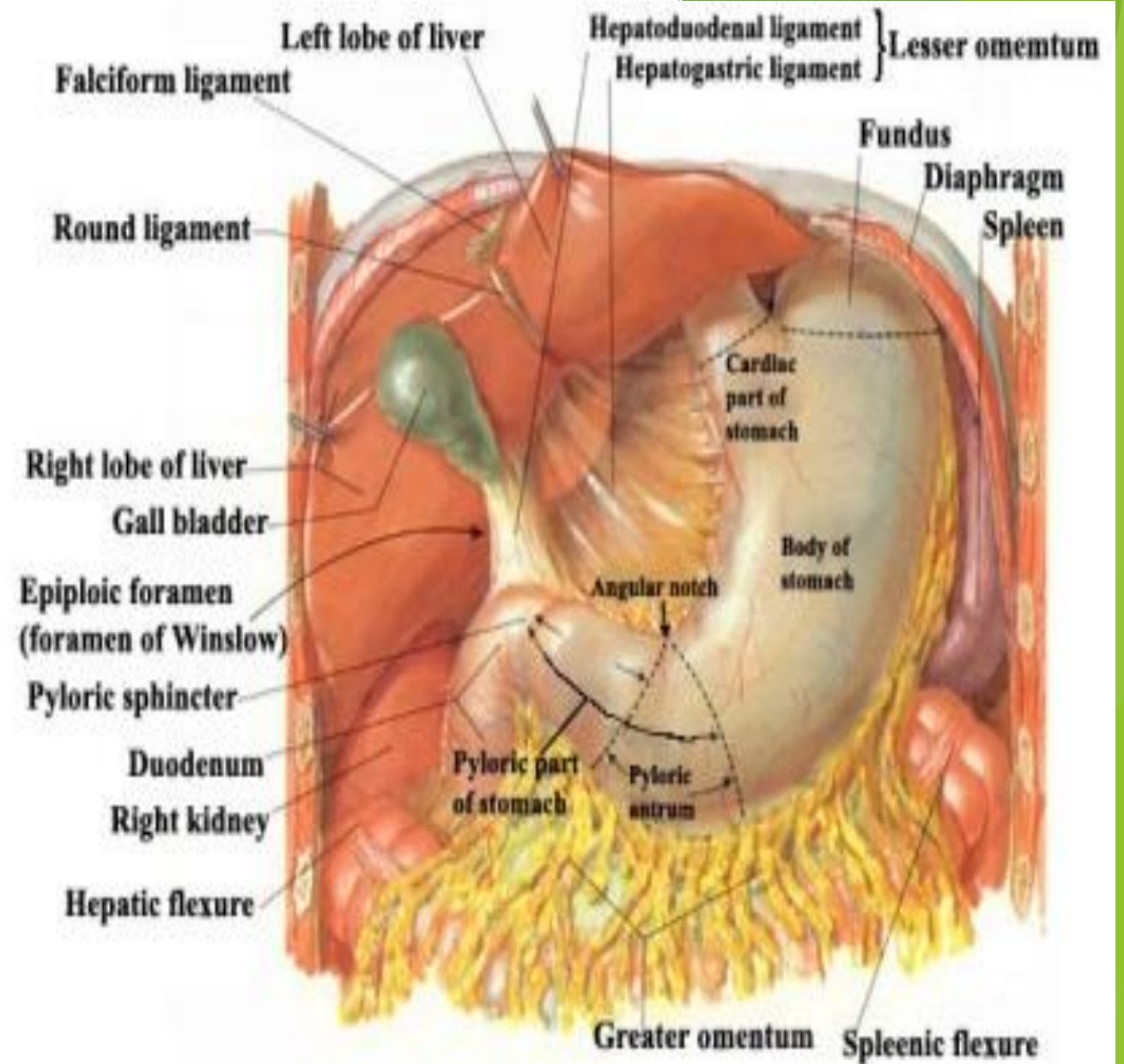
# Parts of the stomach





# The stomach

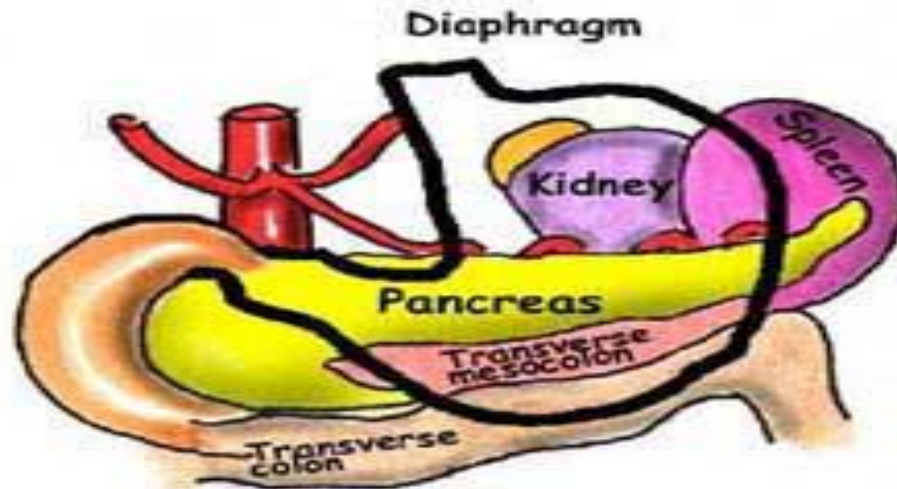
- Layers of peritoneum attached to the stomach:
  - Lesser omentum: attaches the liver to the lesser curvature.
  - Greater omentum: attaches the greater curvature to the posterior body wall.





# Stomach Anatomical Relation

## STOMACH - RELATIONS



### ANTERIOR

Abdominal wall  
Left costal margin  
Diaphragm  
Left lobe of liver

### SUPERIOR

Left dome of diaphragm

### POSTERIOR

Lesser sac  
Pancreas  
Transverse mesocolon  
Transverse colon  
Left kidney/suprarenal gland  
Spleen/splenic artery



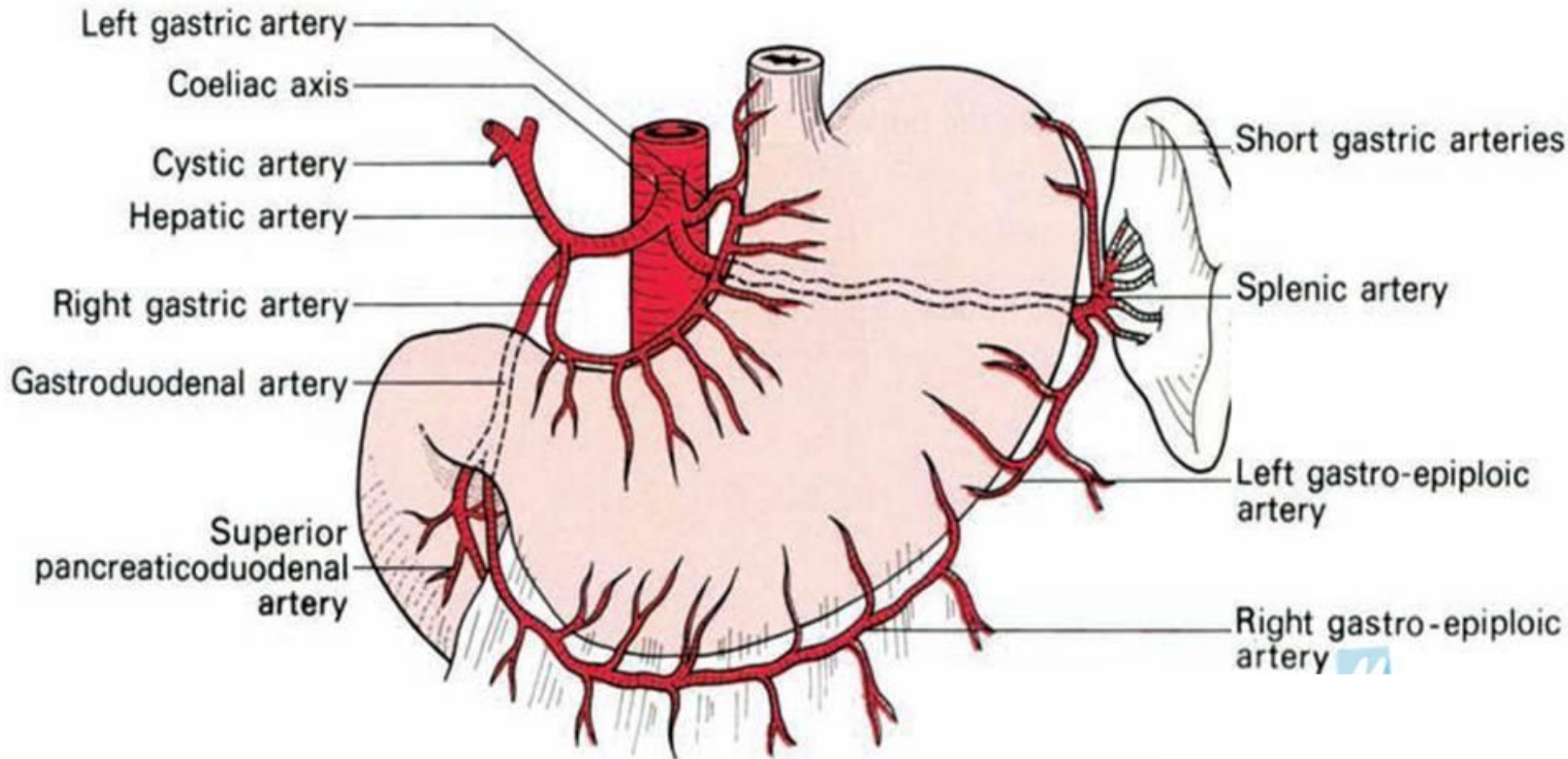
# Stomach Blood Supply

Arterial blood supply:

- ▶ 3 Branches
  - ▶ Left Gastric Artery
    - ▶ Supplies the cardia of the stomach and distal esophagus
  - ▶ Splenic Artery
    - ▶ Gives rise to 2 branches which help supply the greater curvature of the stomach
      - ▶ Left Gastroepiploic
      - ▶ Short Gastric Arteries
  - ▶ Common Hepatic or Proper Hepatic Artery
    - ▶ 2 major branches
      - ▶ Right Gastric- supplies a portion of the lesser curvature
      - ▶ Gastroduodenal artery
        - Gives rise to Right Gastroepiploic artery
        - helps supply greater curvature in conjunction with Left Gastroepiploic Artery

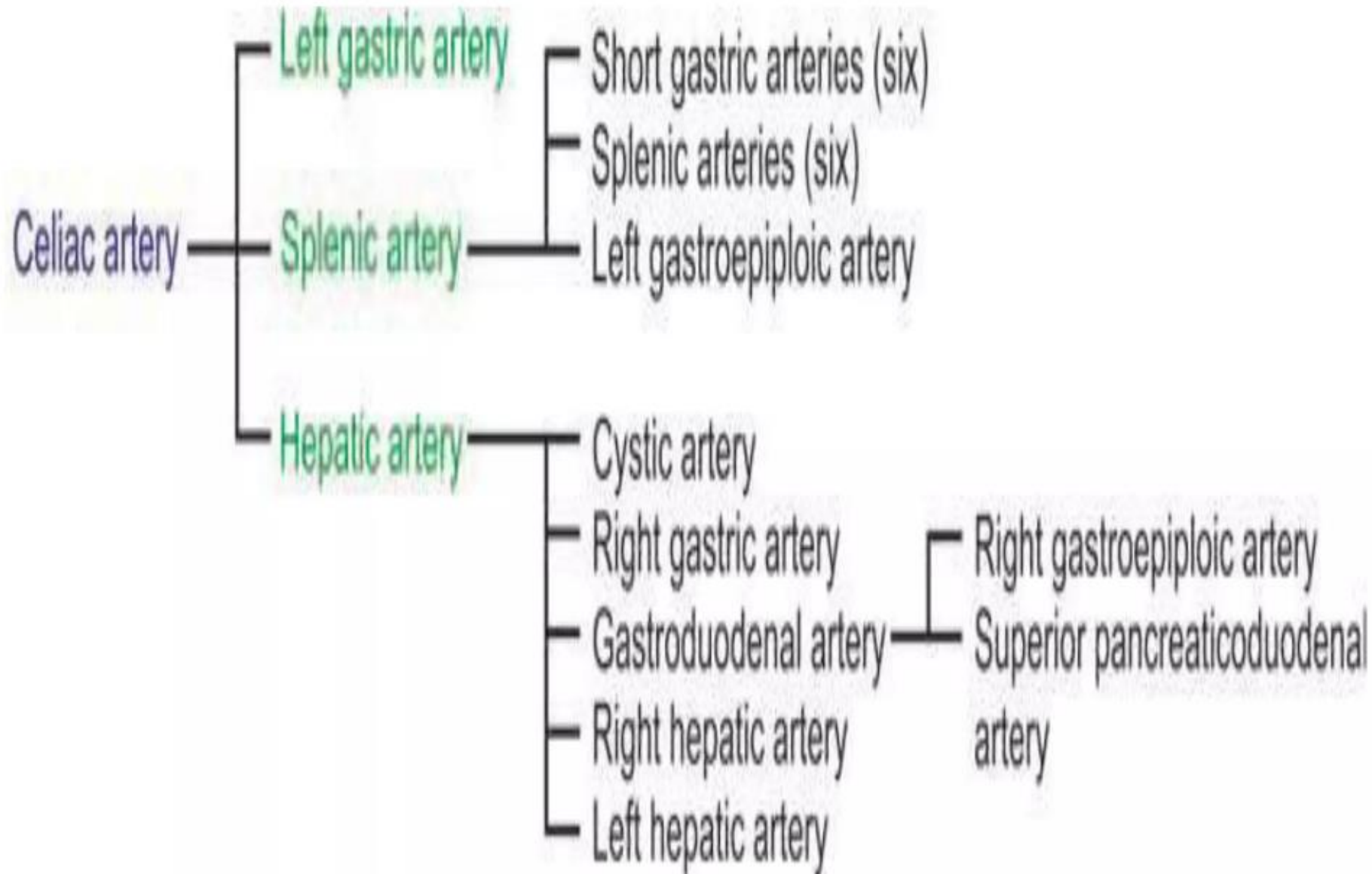


# Blood supply of the Stomach





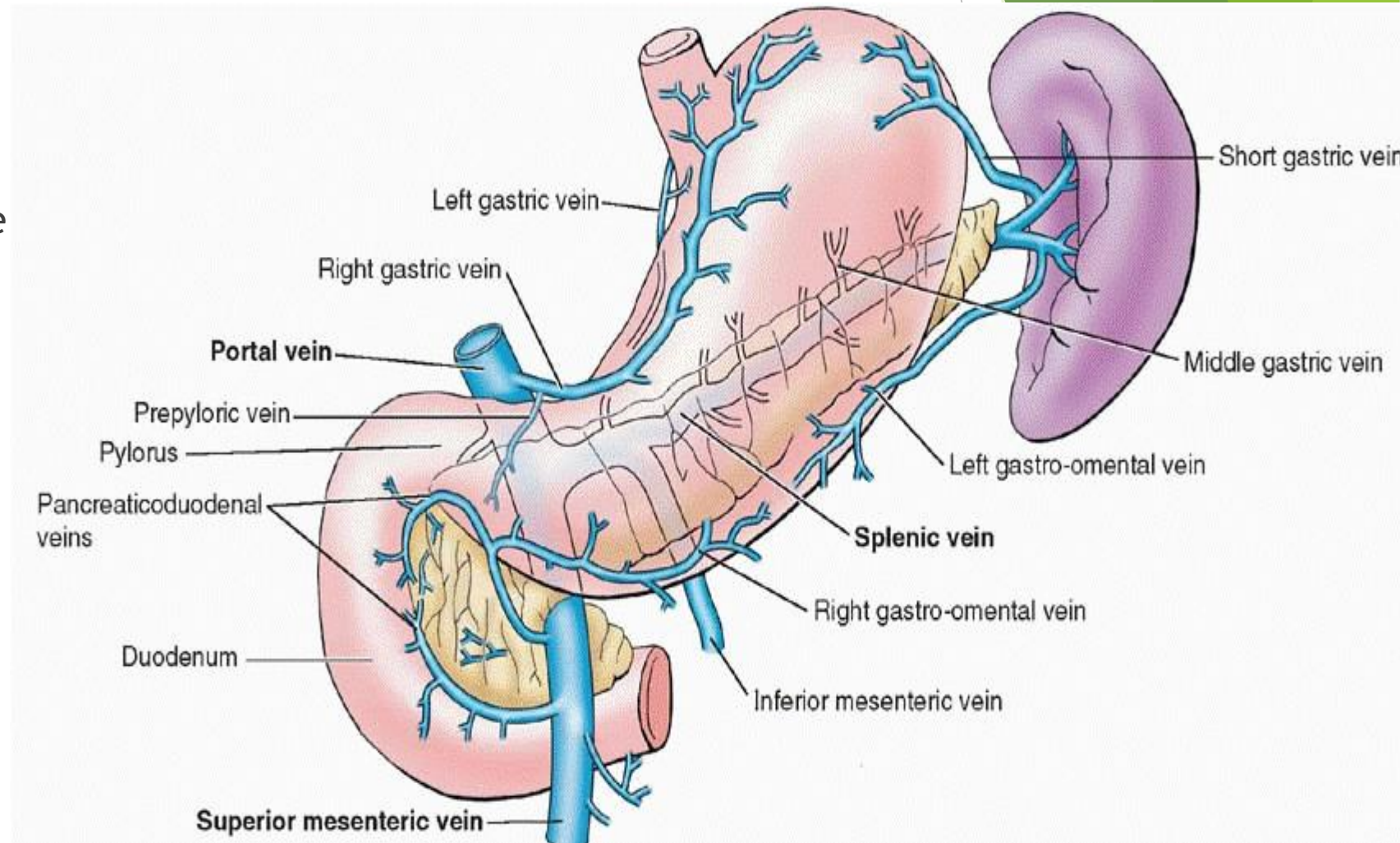
# Arterial Supply Of Stomach





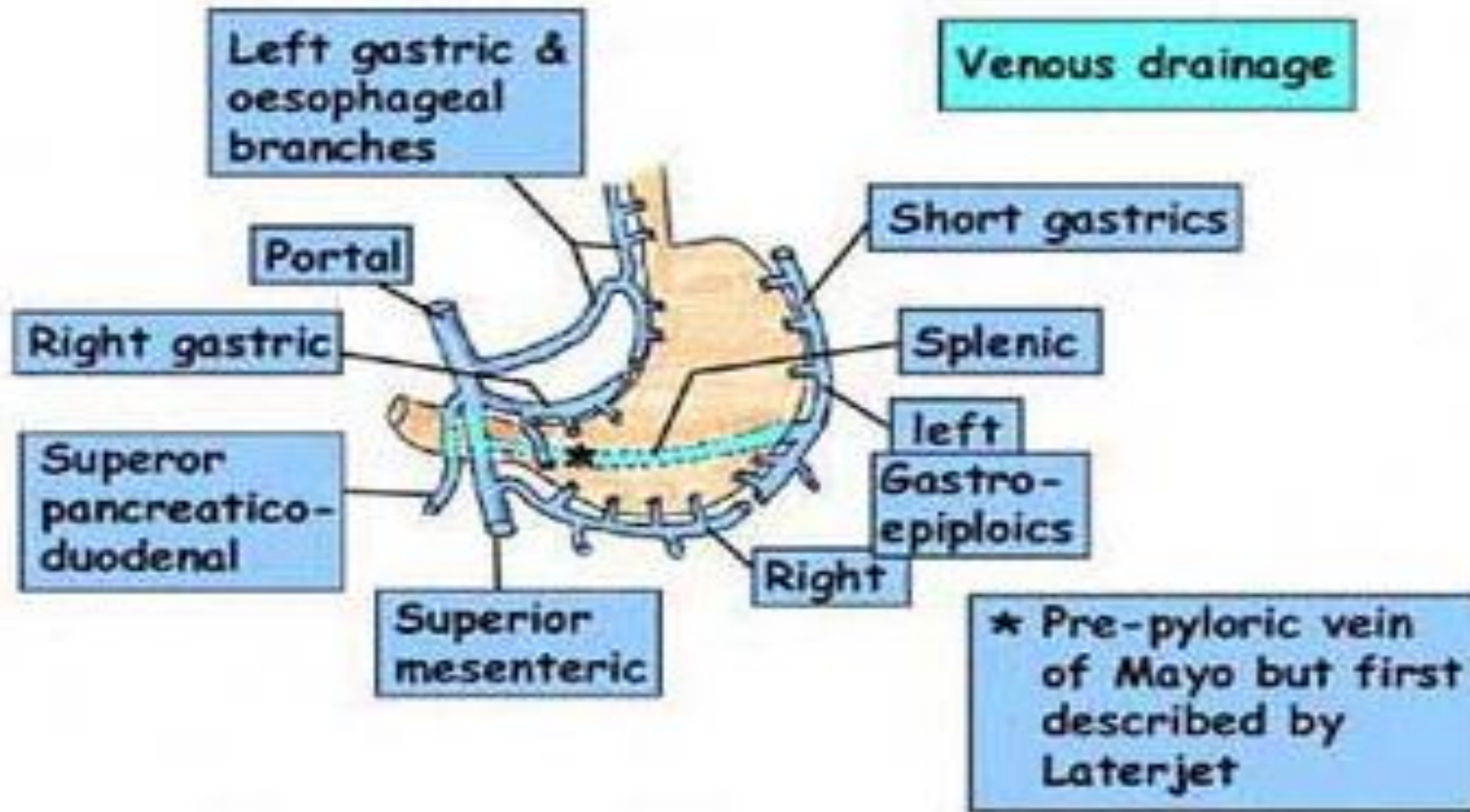
# Stomach Venous Drainage

- ▶ Venous Drainage
  - ▶ Parallel to arterial supply
- ▶ Rt & Lt gastric veins drain to the portal
- ▶ Rt gastroepiploic drains to the SMV
- ▶ Lt gastroepiploic drains to the splenic
- ▶ Short gastric V. into splenic





# Stomach Venous Drainage

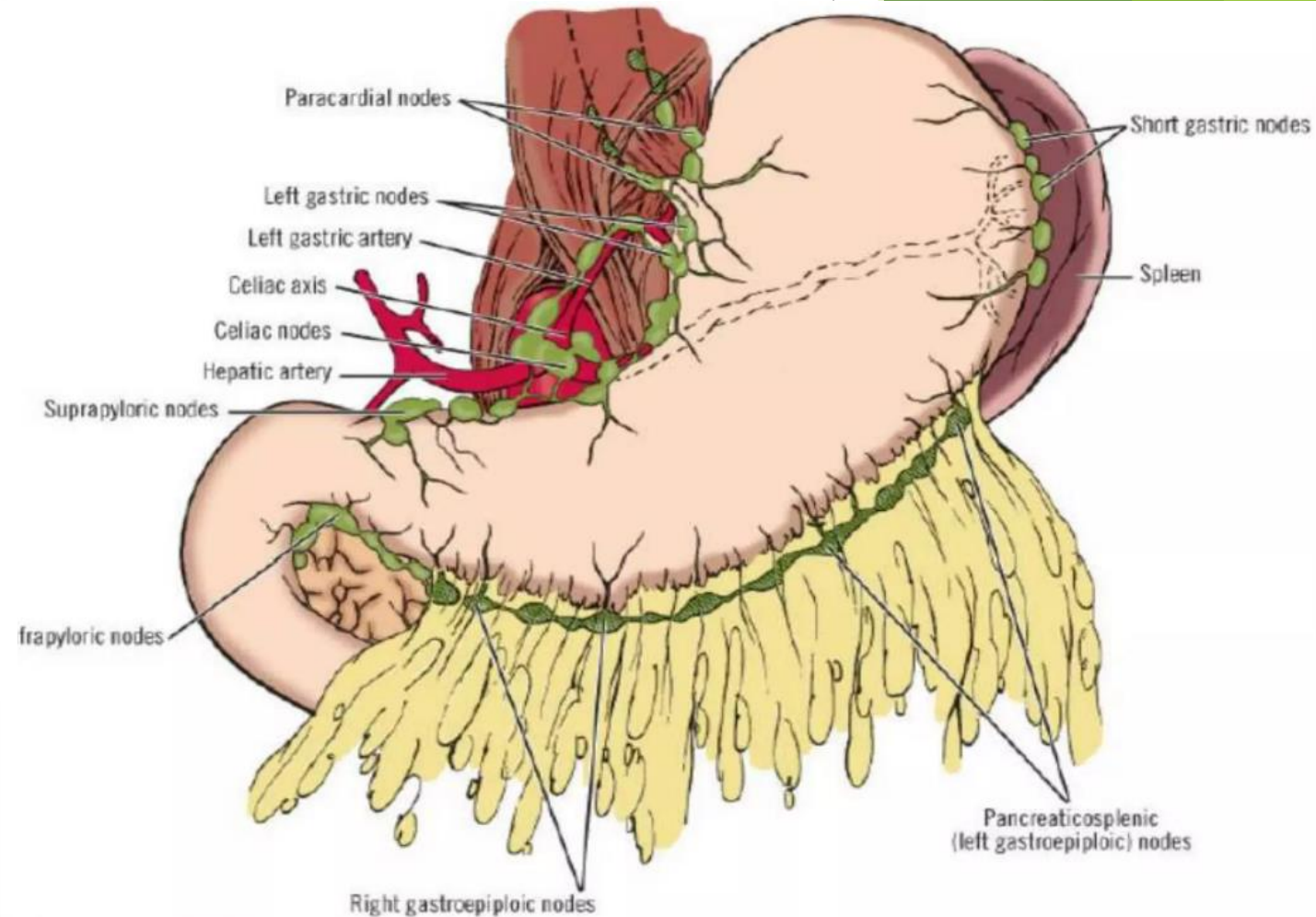




# Stomach Lymphatic Drainage

- Eight groups of lymph nodes of the stomach:

1. Paracardial nodes
2. Left gastric nodes at the left gastric artery
3. Celiac nodes at the celiac artery
4. Suprapyloric nodes
5. Infrapyloric nodes
6. Right gastroepiploic nodes at the pathway of the right gastroepiploic artery
7. Pancreaticosplenic nodes at the pathway of the left gastroepiploic artery
8. Upper greater curvature nodes at the short gastric vessels



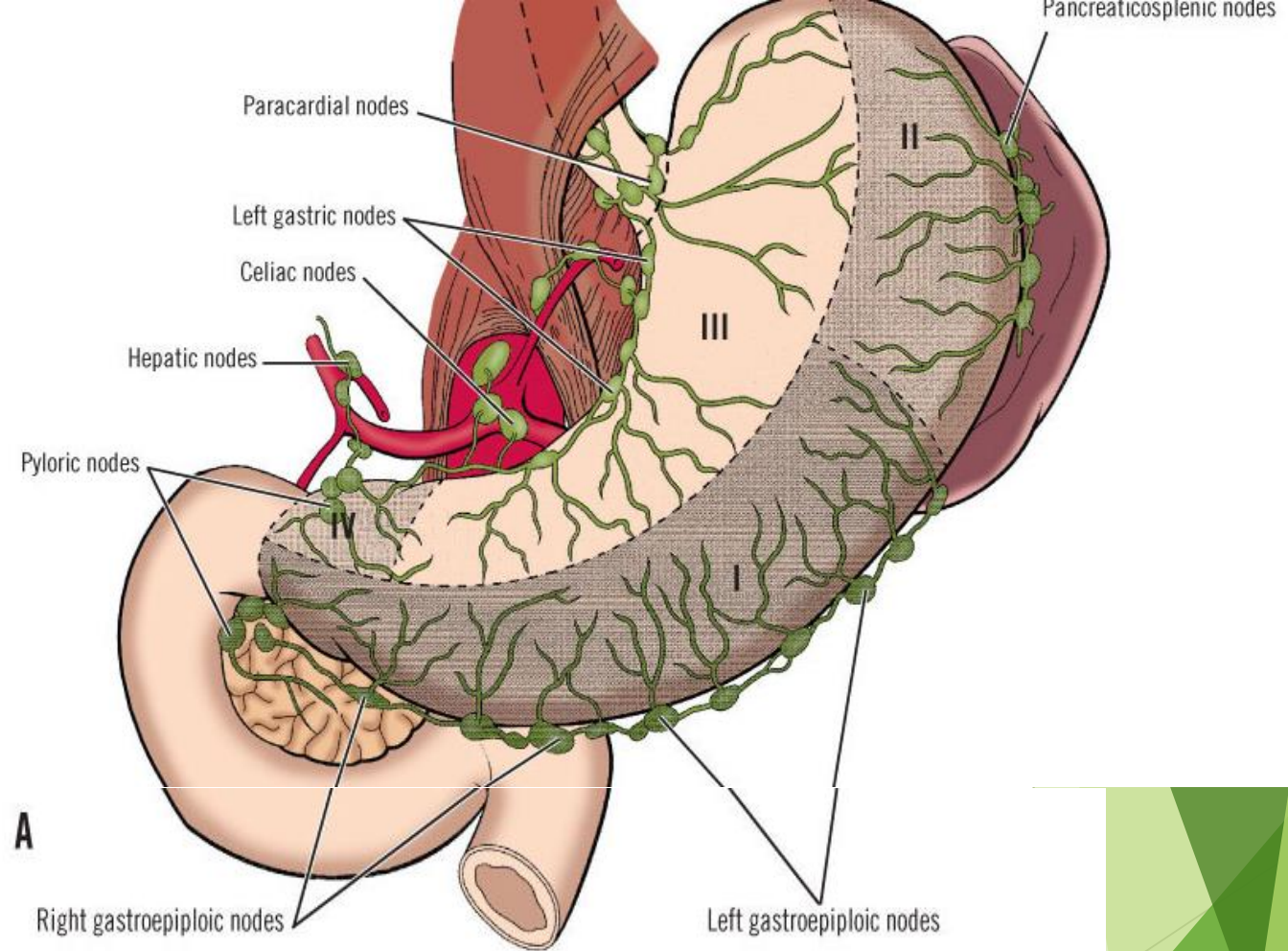
Copyright ©2006 by The McGraw-Hill Companies, Inc.  
All rights reserved.

Eight groups of lymph nodes of the stomach.



1. Left gastric lymph nodes: drains areas of both anterior and posterior gastric walls
2. Pancreaticosplenic nodes: drains gastric fundus and body
3. Right gastroepiploic nodes: drains the right half of the greater curvature, occasionally including the pylorus
4. Hepatic-pyloric-left gastric nodes: drain the pyloric part of the stomach

Zone 1 : left and right gastroepiploic LNs  
 Zone II : Pancreaticosplenic LNs  
 Zone III : left gastric + celiac LNs  
 Zone IV : pyloric + hepatic LNs

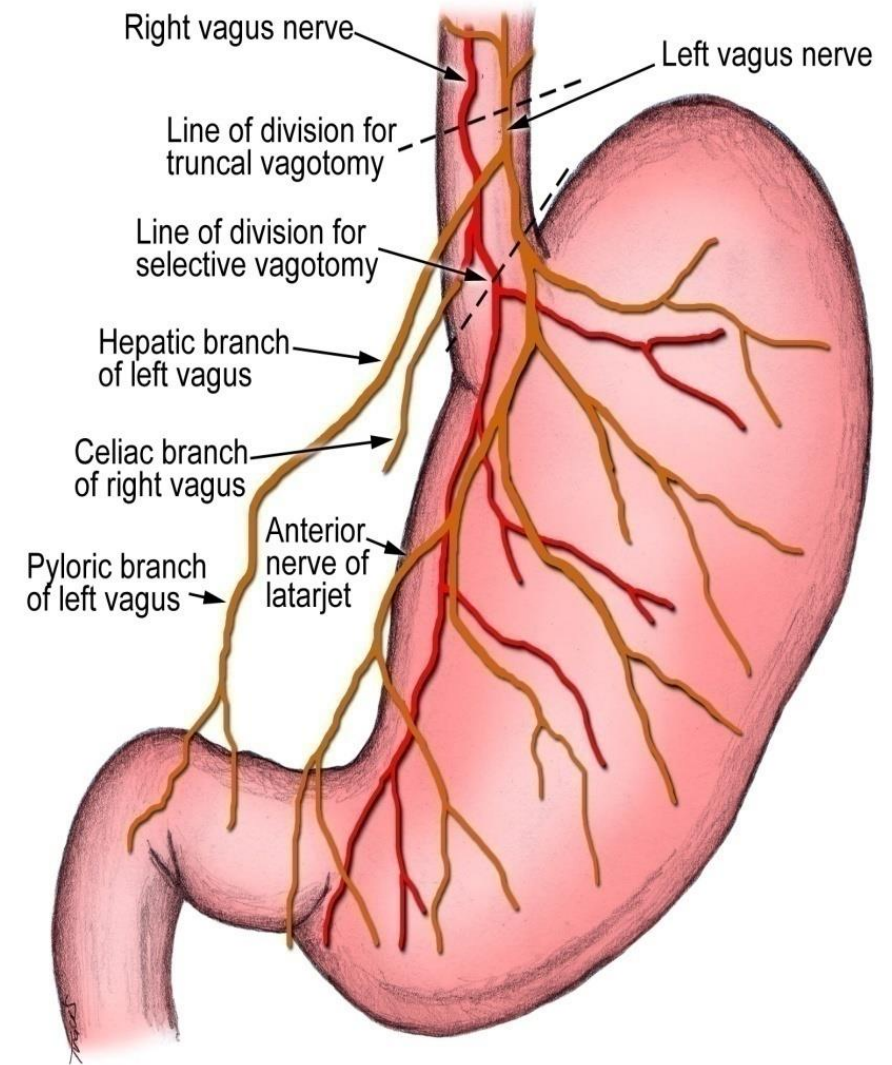


**The 4 zones: I, Inferiorgastric; II, Splenic; III, Superior gastric IV, Hepatic.**



# Stomach Innervations

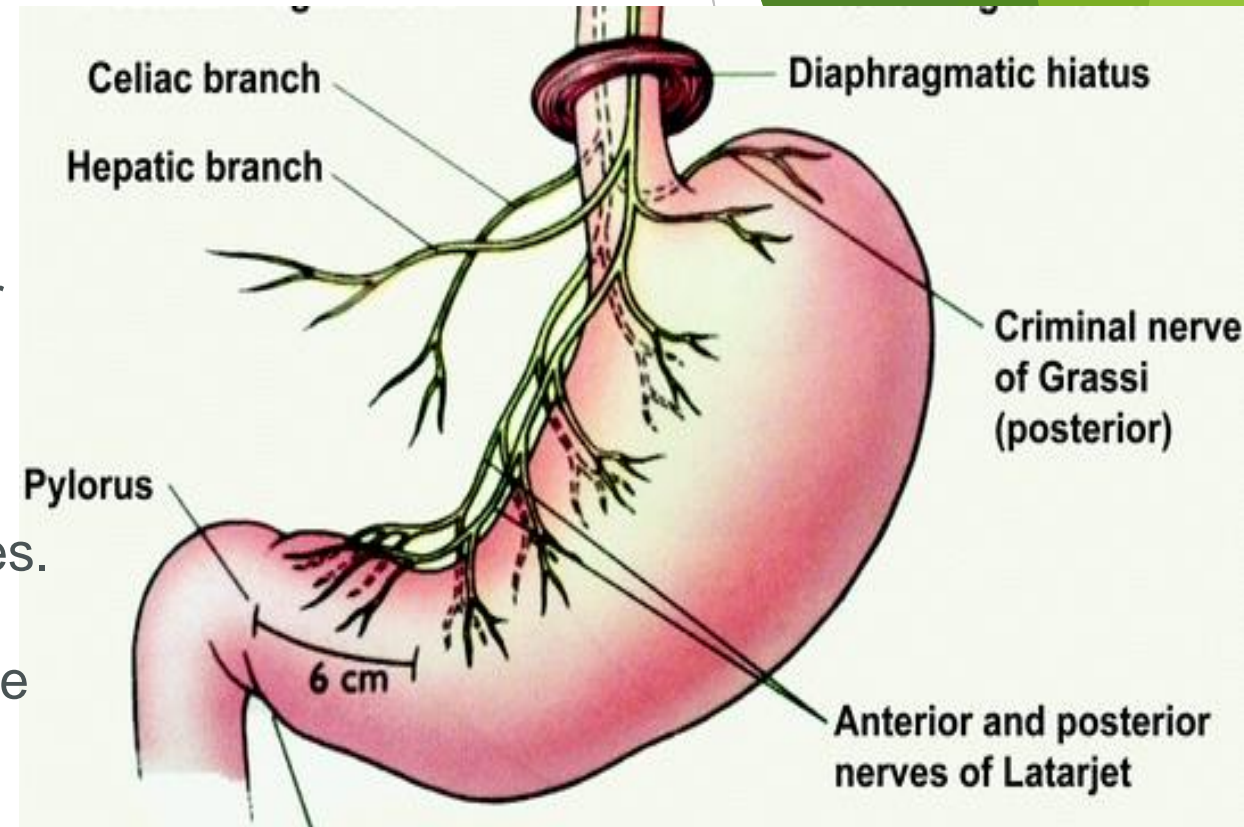
- Parasympathetic innervation of Stomach-Vagus Nerve
- 80% of fibers in vagal trunk is afferent sensory (info transmitting from stomach to CNS) sensations of pain, fullness, and nausea from the stomach.
- **20% motor fibers** for gastric motility and relaxation of the pyloric sphincter during gastric emptying.
- responsible for inducing **gastric acid secretion** also





# Vagus trunk

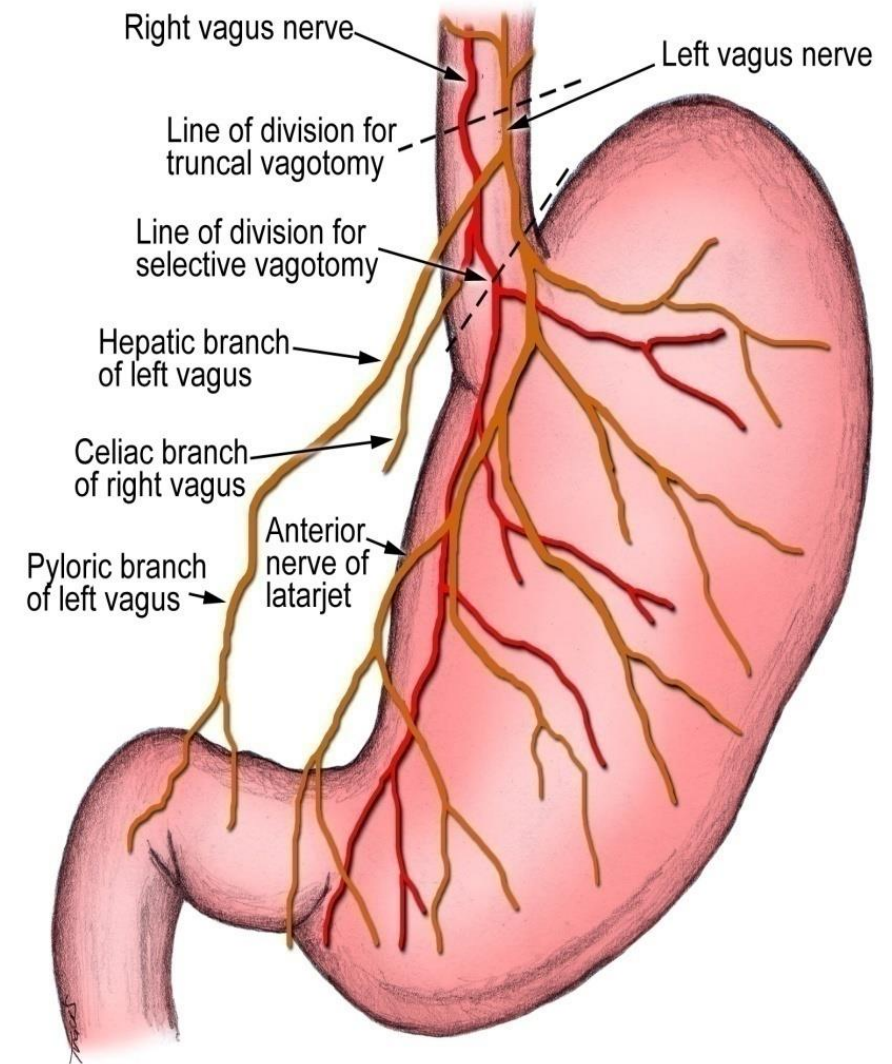
- In the chest → Rt and Lt Vagal trunks
- At the level of the cardia, the left becomes anterior and the right becomes posterior
- The anterior vagal trunk divides into hepatic and anterior gastric (anterior nerve of Latarjet) branches.
- The posterior vagus divides into the posterior nerve of Latarjet and celiac branches.
- **criminal nerve of Grassi** is a proximal branch of the posterior vagal trunk as a possible cause of recurrent ulcers if left undivided during selective vagotomy.





# Levels of vagotomy

- ▶ **Truncal vagotomy** just below the diaphragmatic esophageal hiatus before it gives off celiac and hepatic branches.
- ▶ **selective vagotomy** is performed distal to this location and spares the celiac and hepatic branches.
- ▶ **Highly selective vagotomy** (parietal cell vagotomy) divides individual terminal branches of the nerve of Latarjet in the fundus and corpus of the stomach but spares the vagal branches to the antrum and pylorus, which control gastric motility and emptying—thus spares the need for a drainage procedure.

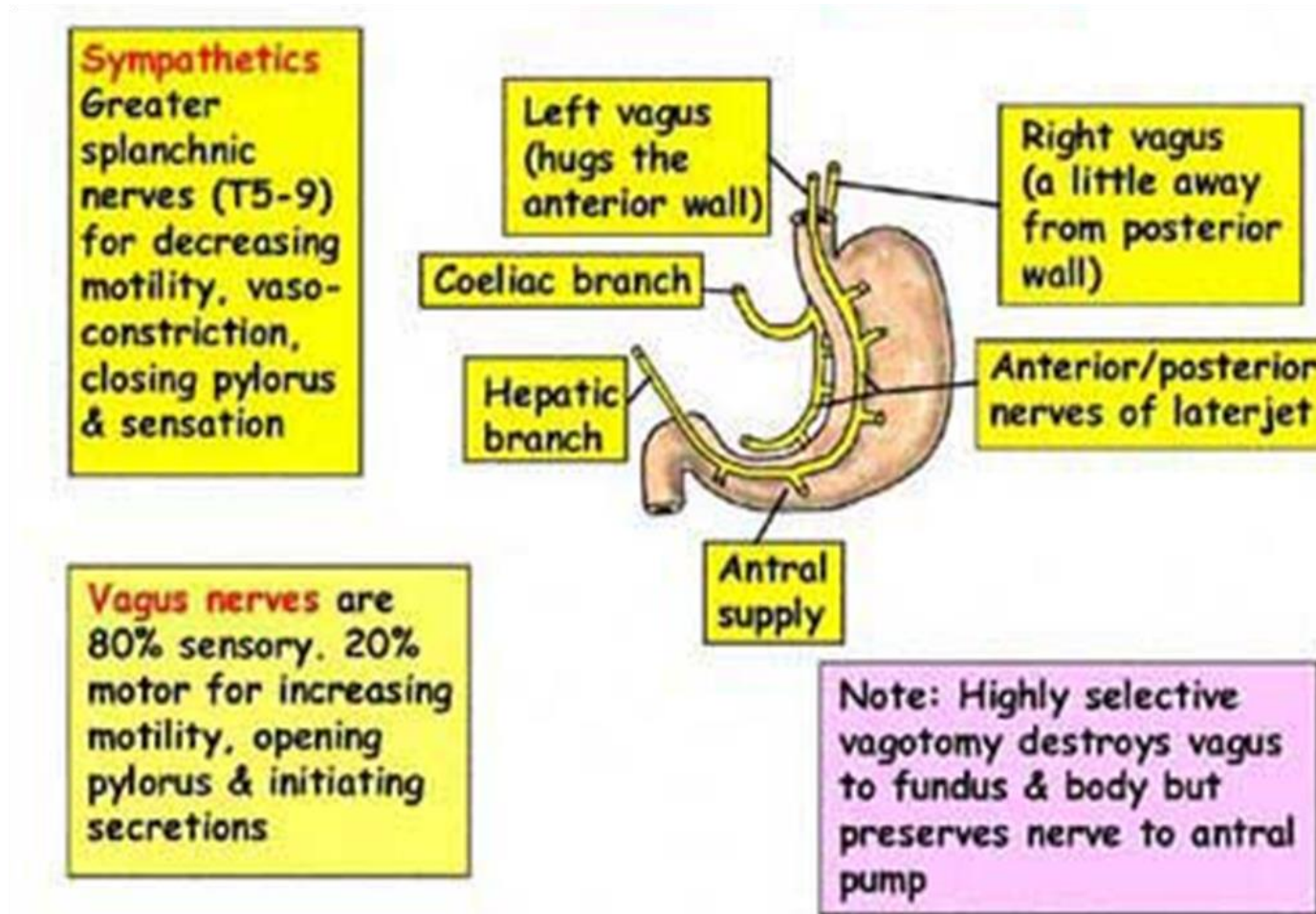




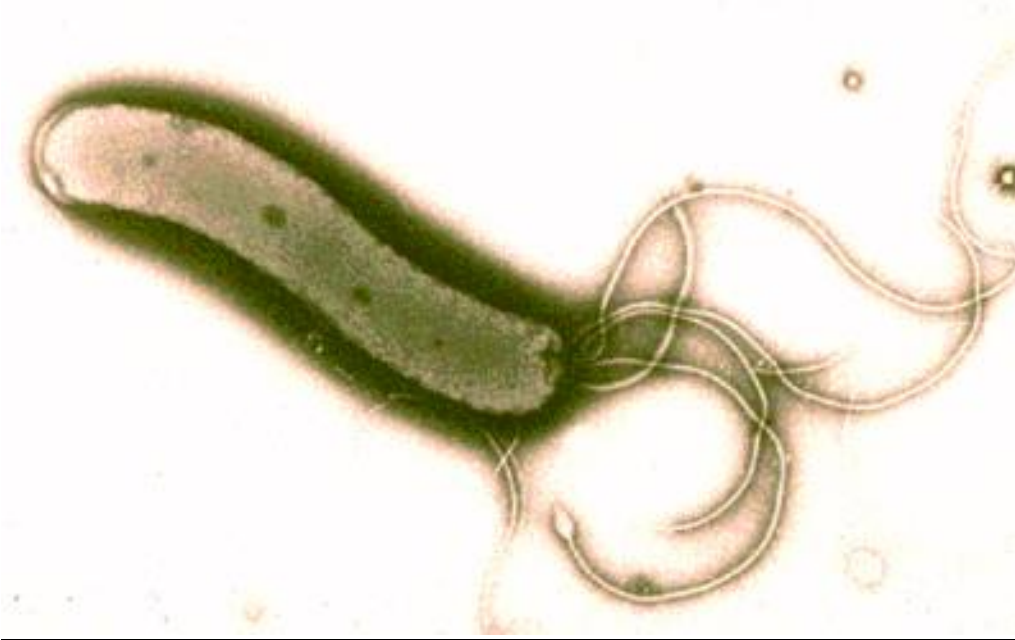
- ▶ **sympathetic** innervation is provided by the celiac plexus from the fifth to twelfth thoracic spinal nerves (T5-T10) and travel to the celiac plexus via the greater splanchnic nerves.
- ▶ Sympathetic innervation is responsible for inhibiting gastric motility and constricting the pyloric sphincter, thus preventing gastric emptying.



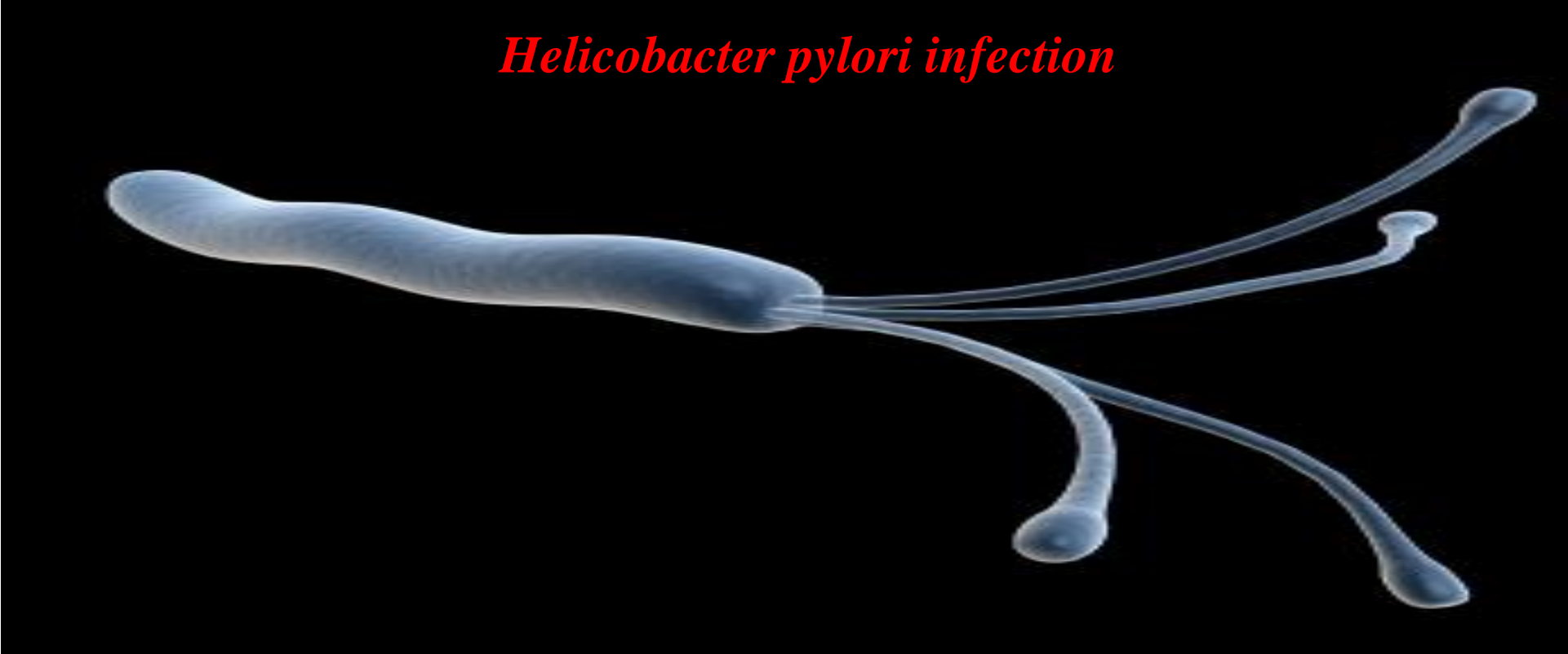
# Stomach Innervations







*Helicobacter pylori infection*





- ▶ H. pylori is a curved or S-shaped, gram-negative microaerophilic motile bacterium whose natural habitat is the human stomach.
- ▶ although **2/3 of population are infected with H. pylori**, only 10 –15% develop PUD & the majority are asymptomatic
- ▶ **Conditions associated with H.Pylori infection :**
- ▶ Acute gastritis → chronic gastritis
- ▶ PUD : 90% of DU , 75% of Gastric Ulcers
- ▶ if Long standing → chronic gastritis → gastric atrophy & increased risk of metaplasia
- ▶ Gastric adenocarcinoma
- ▶ More than 90% of MALT lymphomas have H. pylori & low grade tumours regress with H. pylori eradication only (no need for chemotherapy if responded)



# Mechanisms by Which *H. pylori* can Damage the Gastric Mucosa

Local effects
Elaboration of toxins
vacA
cagA
Effect on immune response
Elaboration of cytokines
Elaboration of interleukin 8
Recruitment of inflammatory cells
Release of inflammatory mediators
Production of immunoglobulins
Effect on acid secretion
Initial hypochlorhydria
Subsequent hyperchlorhydria
Elevated serum gastric levels
Reduced gastric antral somatostatin levels
Increased levels of gastric fundic N-methylhistamine
Hypergastrinemia may contribute to greater parietal cell mass
Effect on duodenal bicarbonate secretion
Reduced secretion of duodenal bicarbonate in patients colonized with <i>H. pylori</i>



- ▶ In patients infected with *H pylori*, high levels of gastrin and pepsinogen and reduced levels of somatostatin have been measured. In infected patients, exposure of the duodenum to acid is increased. Virulence factors produced by *H pylori*, including urease, catalase, vacuolating cytotoxin, and lipopolysaccharide, are all well described.
- ▶ Impair duodenal bicarb secretion which predisposes to DU .



## Pathogenesis

- 1-Increases fasting & postprandial gastrin
- 2-Increases pepsinogen secretion
- 3-Decreases gastric mucosal resistance
- 4-Suppresses somatostatin release
- 5-Releases tissue damaging cytotoxins



# Diagnoses

- ▶ **Noninvasive tests**

- ▶ **urea breath test**

- ▶ analyzes breath for labeled carbon dioxide produced by bacterial urease from the conversion of ingested labeled urea. Because of its noninvasiveness plus high sensitivity and specificity (95%), the urea breath test is considered the test of choice for screening and for documentation of *H. pylori* eradication.

- ▶ **serology.** (serum antibodies, fecal antigen)

- ▶ Serologic tests are quick and inexpensive but cannot differentiate between active infection and previous exposure. Serology is useful for the initial diagnosis of *H. pylori* infection in patients in whom endoscopy is not indicated.



- ▶ **Invasive tests** require endoscopic mucosal biopsy and include histologic examination, the rapid urease test, and culture.
- ▶ **Histologic examination** can accurately diagnose *H. pylori* with two biopsy specimens with high sensitivity and specificity (90%).
- ▶ **The rapid urease test** on a mucosal biopsy is considered the initial test of choice because of its simplicity, accuracy, and rapid results.
- ▶ **Culture of *H. pylori*** has the most specificity (100%) but is difficult to perform (only for research purposes or patients with suspected antibiotic resistance).



# Treatment : Eradication therapy

A) P.P.I based triple therapy with two antibiotic

B) H2 blockers based triple therapy

\*Bismuth is included in some regimens

Table 1. First-line treatment recommendations

Therapy	Dosing	Duration (days)	Eradication (%)
<b>Clarithromycin triple</b>	PPI twice daily + clarithromycin 500 mg twice daily + amoxicillin 1 g twice daily or metronidazole 500 mg three times daily	14	70%-85%
<b>Bismuth quadruple</b>	PPI twice daily + bismuth salicylate 300 mg four times daily + metronidazole 500 mg three times daily + tetracycline 500 mg four times daily	14	75%-90%
<b>Non-bismuth quadruple</b>	PPI twice daily + amoxicillin 1 g twice daily + clarithromycin 500 mg twice daily + metronidazole 500 mg three times daily	14	90%

PPI, proton pump inhibitor



# Peptic ulcer disease : PUD



- ▶ **Peptic ulcers** are defects in the gastric or duodenal mucosa that extend through the muscularis mucosa.
- ▶ Loss of normal balance between protective mechanisms and acid secretion/aggressive factors
- ▶ Normal protective mechanisms :
  - tight intercellular junctions, mucus production, bicarbonate, mucosal blood flow, cellular restitution, Prostaglandin E , and epithelial renewal.
  - Risk factors :
    - *H pylori* infection
    - Drugs (NSAIDS, steroids)
    - Severe physiologic stress (burns/curling ulcer, head trauma/ cushing ulcer.... etc
    - Hypersecretory states (uncommon) – Zollinger Ellison , G-cell hyperplasia
    - Genetic factors
    - smoking
- ▶ Incidence of PUD is decreasing due to H.pylori eradication and anti secretory therapy



## Differential Diagnosis of Hypergastrinemia

With excessive gastric acid formation (ulcerogenic)

Zollinger-Ellison syndrome

Gastric outlet obstruction

Retained gastric antrum (after Billroth II reconstruction)

G-cell hyperplasia

Without excessive gastric acid formation (nonulcerogenic)

Pernicious anemia

Atrophic gastritis

Renal failure

Postvagotomy

Short gut syndrome (after significant intestinal resection)



# History and Exam

- ▶ Epigastric pain, (gnawing or burning)
  - Dyspepsia, including belching, bloating, distention, and fatty food intolerance
  - Heartburn
  - Chest discomfort
  - Hematemesis or melena.
  - anemia (eg, fatigue, dyspnea)
  - Sudden onset of symptoms may indicate perforation.
  - NSAID-induced gastritis or ulcers may be silent, especially in elderly patients.
- ▶ On exam : normal or minimal findings if uncomplicated (minimal epigastric tenderness)
- ▶ Signs of GOO (if strictures and obstruction) or peritonitis (if perforation occurred)



	<u><b>Duodenal Ulcer</b></u>	<u><b>Gastric ulcer</b></u>
<b>Relived by</b>	Eating	Lying down or vomiting
<b>Duration</b>	1-2 months	Few weeks
<b>Vomiting</b>	Uncommon	Common(to relieve the pain)
<b>Appetite</b>	Good	Pt. afraid to eat
<b>Diet</b>	Good , eat to relieve the pain	Avoid fried food
<b>Weight</b>	No wt. loss	wt. Loss maybe present
<b>At night</b>	Pain awaken pt	Less night pain



# Alarming symptoms :

- Relentless pain
- Bleeding or anemia
- Early satiety
- Unexplained weight loss
- Progressive dysphagia
- Recurrent vomiting
- Family history of gastrointestinal cancer
- → all need endoscopy + biopsy to be performed



# workup

- ▶ H.pylori testing
- ▶ Endoscopy + biopsy → for all suspected PUD especially if alarming symptoms present or new onset/refractory symptoms in >50 years old patients.
- ▶ Erect CXR (if perforation suspected)
- ▶ Serum gastrin level (if multiple, unusual location, +FHx, unidentifiable cause, +diarrhea,+steatorrhea,weight loss, ulcer refractory to treatment) → to exclude ZES
- ▶ Secritin stimulation test (for gastrinoma / ZES)
- ▶ Angiography+ Embolization (if massive bleeding)



# Treatment :

- ▶ H.pylori eradication (triple therapy)
- ▶ Confirm eradication of H.pylori and healing of ulcer with followup endoscopy
- ▶ Stop NSAIDs, PGE analogue (misoprostol) , prophylactic PPI
- ▶ SURGICAL treatment :
  - Now largely abandoned because of high success rate for medical therapy.
  - Surgery is indicated only in refractory ulcers not responding to medical treatment (12weeks)
  - or if complications developed (bleeding not responding to endoscopic management)
  - Perforation, GOO , Suspicion of malignancy



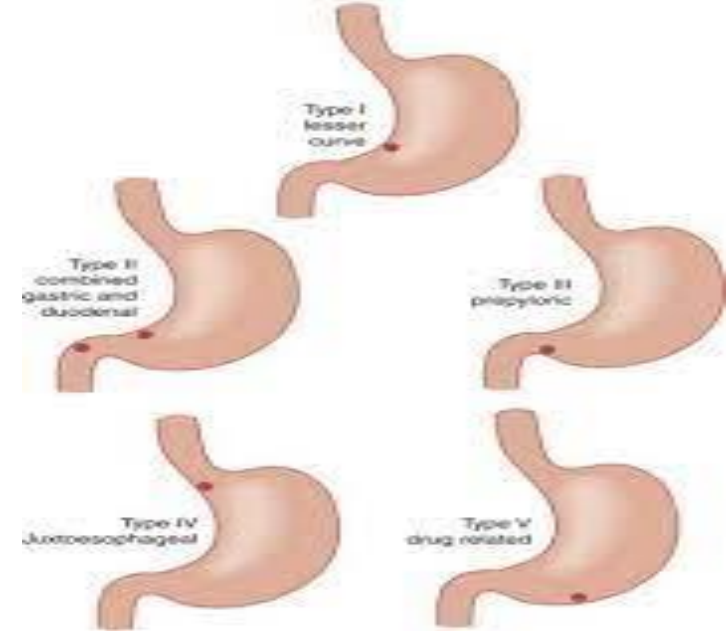
# Modify Johnson's classification

Type	Location	Acid Hypersecretion
I	Lesser curvature, incisura	No
II	Body of stomach, incisura, and duodenal ulcer (active or healed)	Yes
III	Prepyloric	Yes
IV	High on lesser curve, near gastroesophageal junction	No
V	Anywhere (medication induced)	No

Antrectomy only

Type II,III treated as duodenal ulcer, preferred tx include antrectomy (to include ulcer) + truncal vagotomy with Billroth I OR II

Modified Antrectomy including ulcer+ billroth I OR subtotal gastrectomy with Roux en Y without vagotomy





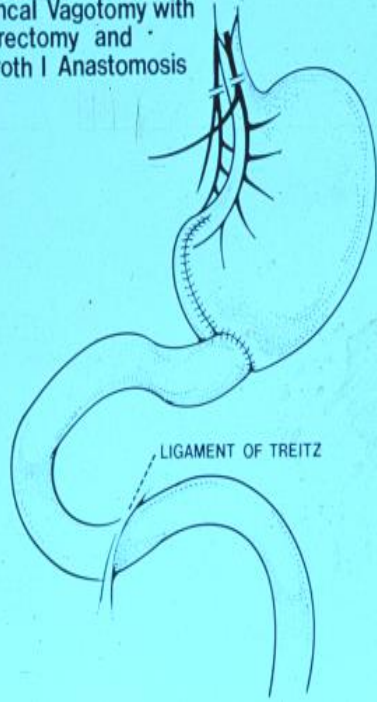
# SURGICAL OPTION

- VAGOTOMY
  - Truncal and drainage
  - Selective
  - Highly selective
  - Posterior vagotomy and anterior seromyotomy
- GASTRECTOMY
  - Billroth I
  - Billroth II
  - Subtotal gastrectomy
- GRAHAM'S OMENTAL PATCH
- SUTURE LIGATION OF GASTRODUODENAL ARTERY
- UNDER-RUNNING AN ULCER BASE
  - After excision of the edge
  - Vagotomy

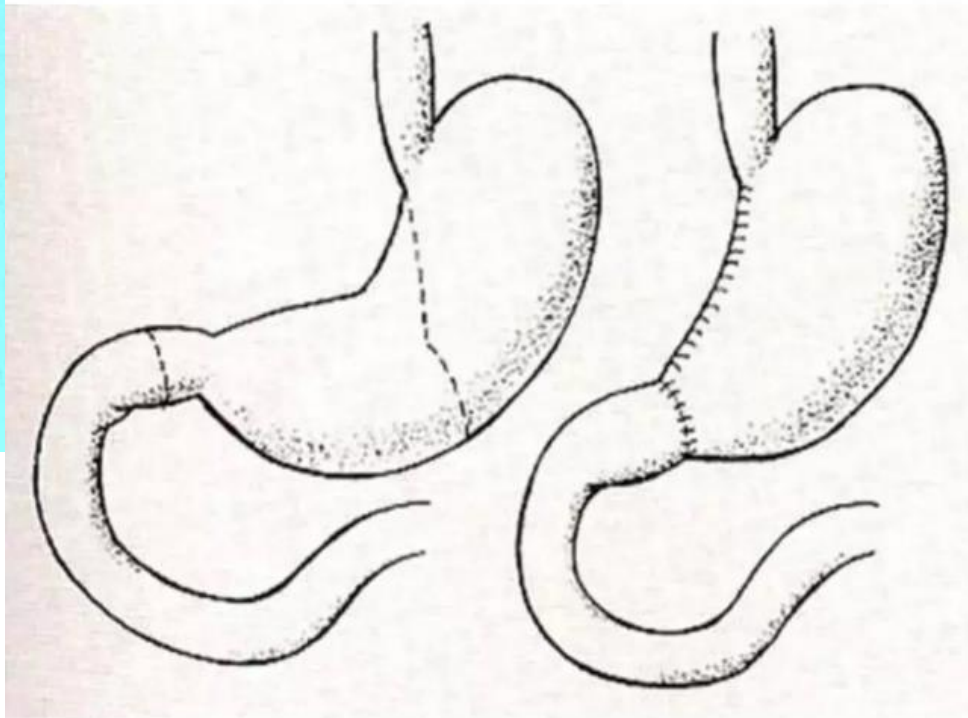


# Billroth I

Truncal Vagotomy with  
Antrectomy and  
Billroth I Anastomosis



**Billroth I – partial gastrectomy gastro-duodenostomy end-to-end**  
**Done for gastric ulcer in the antrum**



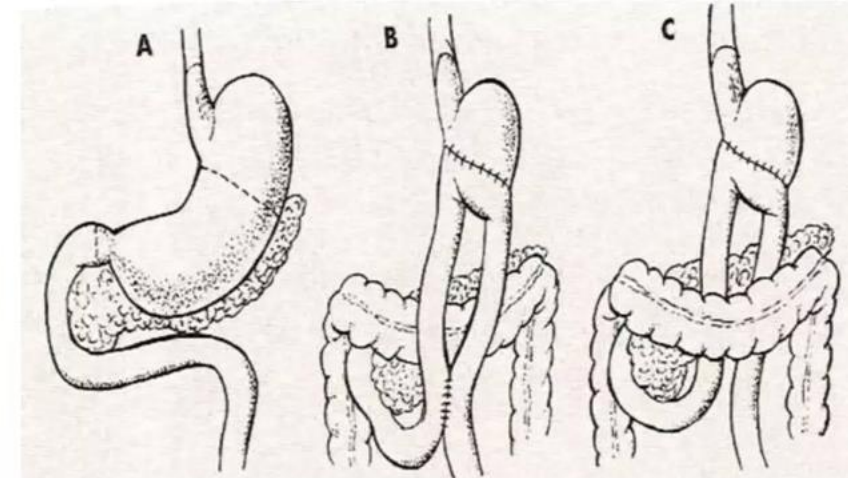
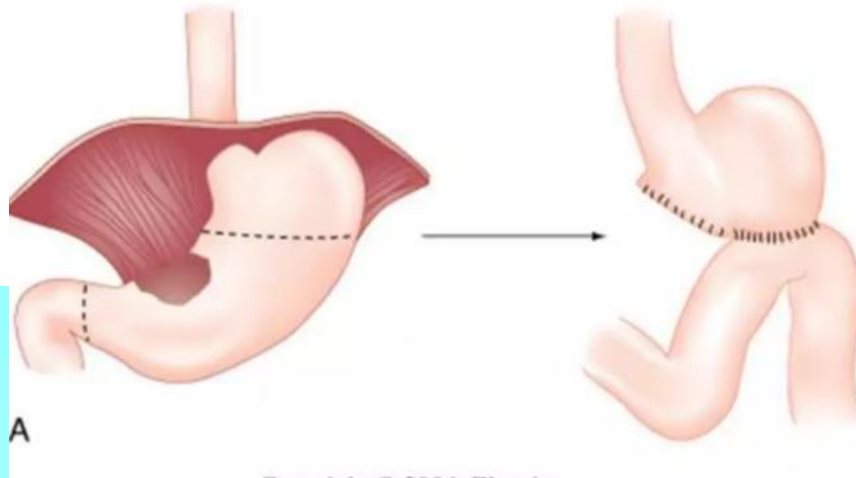
Billroth I gastroduodenal  
anastomosis completed



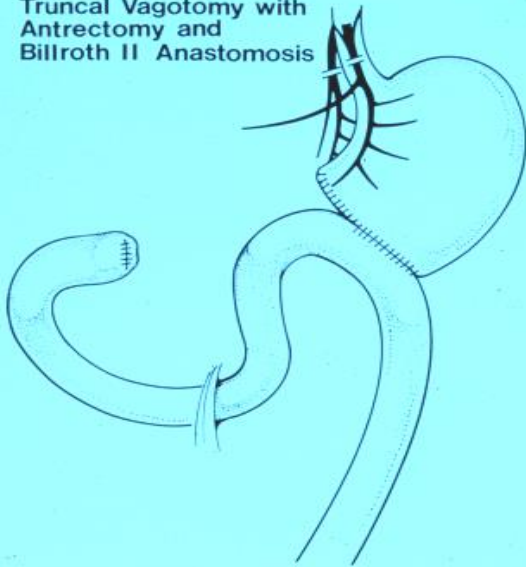
# Billroth II

Partial gastro-jejunostomy end-to-side with blind closure of duodenum

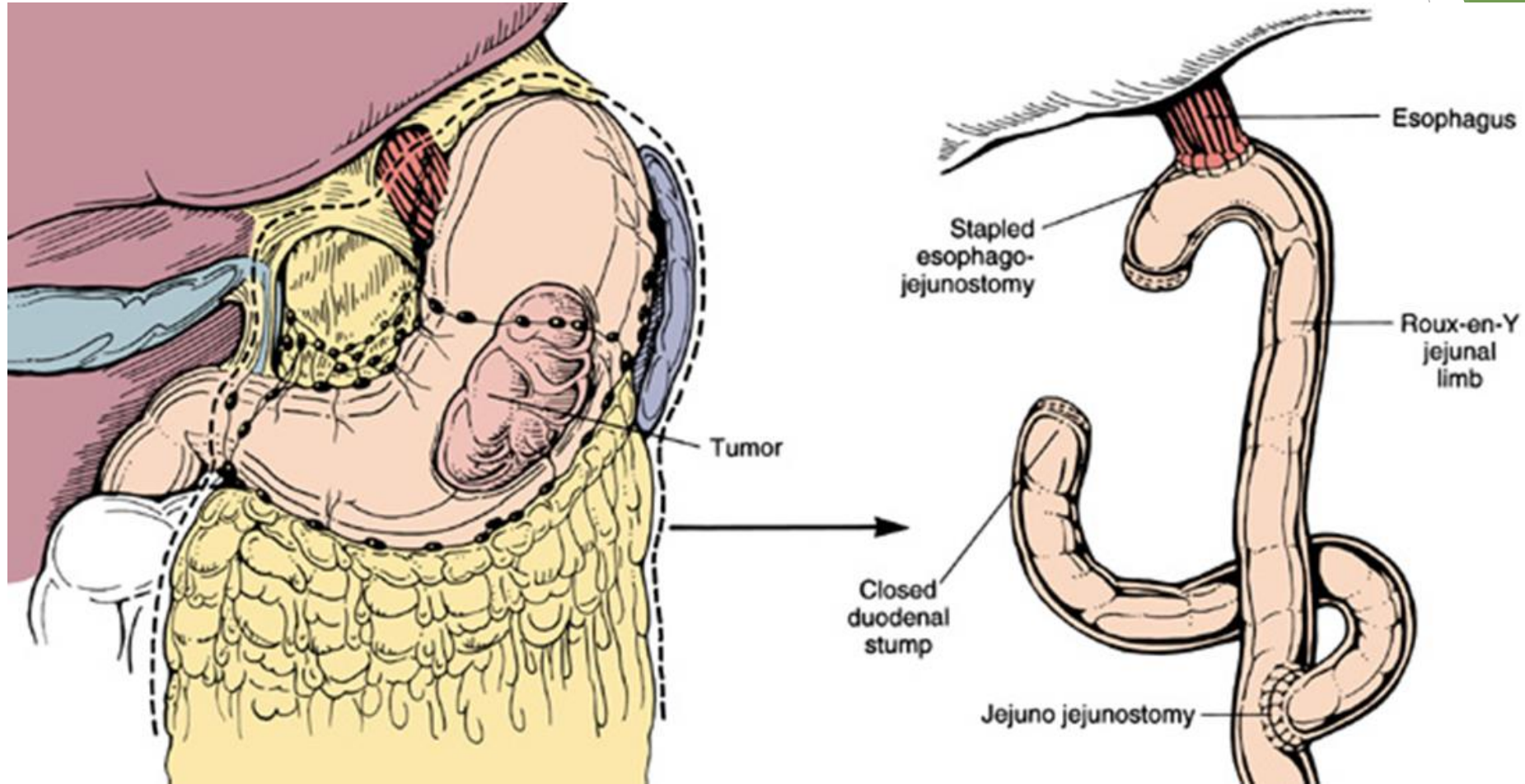
Done for a proximal gastric ulcer



Truncal Vagotomy with  
Antrectomy and  
Billroth II Anastomosis









**Table 25-12 Surgical Options in the Treatment of Duodenal and Gastric Ulcer Disease**

Indication	Duodenal	Gastric
Bleeding	<ol style="list-style-type: none"><li>1. Oversew<sup>a</sup></li><li>2. Oversew, V+D</li><li>3. V+A</li></ol>	<ol style="list-style-type: none"><li>1. Oversew and biopsy<sup>a</sup></li><li>2. Oversew, biopsy, V+D</li><li>3. Distal gastrectomy<sup>b</sup></li></ol>
Perforation	<ol style="list-style-type: none"><li>1. Patch<sup>a</sup></li><li>2. Patch, HSV<sup>b</sup></li><li>3. Patch, V+D</li></ol>	<ol style="list-style-type: none"><li>1. Biopsy and patch<sup>a</sup></li><li>2. Wedge excision, V+D</li><li>3. Distal gastrectomy<sup>b</sup></li></ol>
Obstruction	<ol style="list-style-type: none"><li>1. HSV + GJ</li><li>2. V+A</li></ol>	<ol style="list-style-type: none"><li>1. Biopsy; HSV + GJ</li><li>2. Distal gastrectomy<sup>b</sup></li></ol>
Intractability/nonhealing	<ol style="list-style-type: none"><li>1. HSV<sup>b</sup></li><li>2. V+D</li><li>3. V+A</li></ol>	<ol style="list-style-type: none"><li>1. HSV and wedge excision</li><li>2. Distal gastrectomy</li></ol>

<sup>a</sup>Unless the patient is in shock or moribund, a definitive procedure should be considered.

<sup>b</sup>Operation of choice in low-risk patient.

V : vagotomy

HSV: highly selective vagotomy

D: drainage pyloroplasty

A: antrectomy

GJ: gastrojejunostomy



Duodenal	Highly-selective anterior vagotomy combined with posterior truncal vagotomy or seromyotomy combined with posterior truncal vagotomy
Gastric	Type I: Distal gastrectomy Type II: Antrectomy with vagotomy Type III: Antrectomy with vagotomy Type IV: Subtotal gastrectomy
Uncertain diagnosis*	The same than in case of refractory ulcer
Gastric outlet obstruction*	Highly-selective vagotomy with gastrojejunostomy (if balloon dilatation fails)
Bleeding duodenal ulcer	Partial gastrectomy (less rebleeding rate) Suture oversewing (less long-term side effects)
Bleeding gastric ulcer	Ulcer excision (variable from wedge excision to partial gastrectomy)
Perforated duodenal ulcer	Simple closure Acid secretion reduction procedure if continuation of NSAIDs is predictable
Perforated gastric ulcer	Partial gastrectomy Biopsy and simple closure in case of patient poor condition

**Table 2** Indications for surgery and recommended techniques \*After excluding diseases other than peptic



## For duodenal ulcers :

- Acid can be reduced by eliminating vagal stimulation, removing the antral source of gastrin, and removing the parietal cell mass.
- subtotal two-thirds gastrectomy has the highest mortality rate.(abandoned)
- Truncal vagotomy with antrectomy has the lowest recurrence rate.
- Procedures involving antrectomy, pyloroplasty, or truncal vagotomy may be complicated by diarrhea, postprandial dumping, or bile reflux.
- Selective vagotomy, which preserves the hepatic and celiac vagal branches, has been associated with a lower rate of diarrhea than truncal vagotomy has.
- Highly selective vagotomy, preserve innervations to the pyloro-antral region and thus maintain more normal gastric emptying. This operation carries the lowest mortality rate, the lowest incidence of side effects (morbidity) , but the highest recurrence rate, which ranges from 5% to 15%.



**Table 25-13 Clinical Results of Surgery for Duodenal Ulcer**

	<b>Parietal Cell Vagotomy</b>	<b>Truncal Vagotomy and Pyloroplasty</b>	<b>Truncal Vagotomy and Antrectomy</b>
Operative mortality rate (%)	0	<1	1
Ulcer recurrence rate (%)	5-15	5-15	<2
Dumping (%)			
Mild	<5	10	10-15
Severe	0	1	1-2
Diarrhea (%)			
Mild	<5	25	20
Severe	0	2	1-2

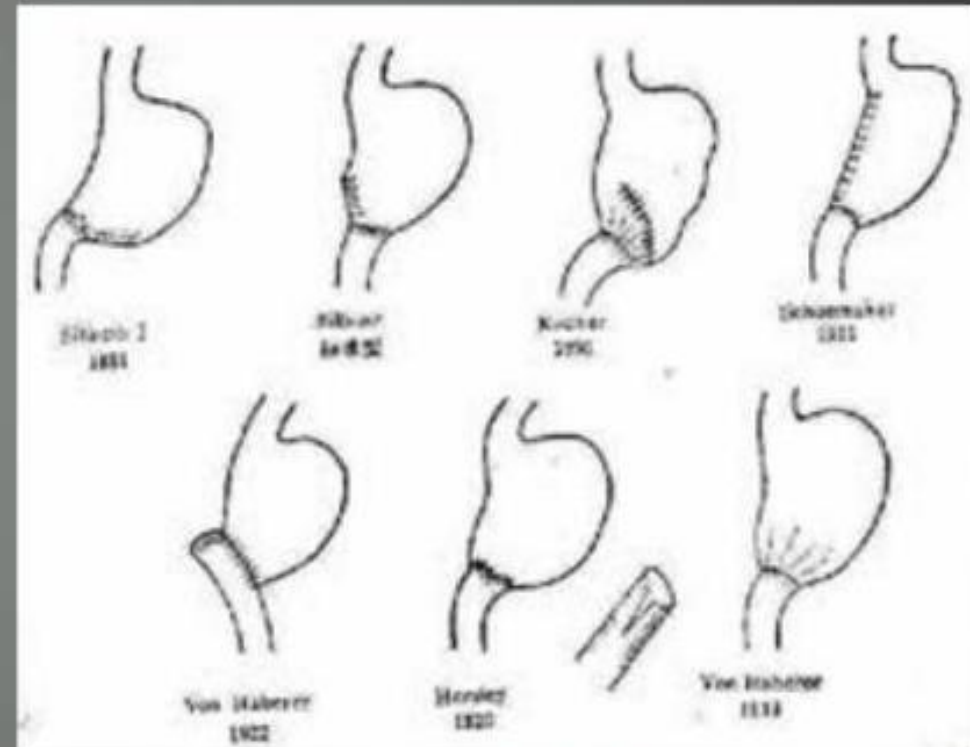
SOURCE: Modified with permission from Mulholland MW, Debas HT: Chronic duodenal and gastric ulcer. *Surg Clin North Am* 67:489, 1987.



# Billroth I

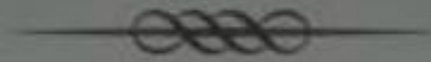


- Preserves duodenal passage
- Preserves pancreatic function
  - Altered after gastrojejunostomy
  - Better fat digestion
- Less incidence of gastritis and reflux
- Can use only with distal limited gastric resections
- tension

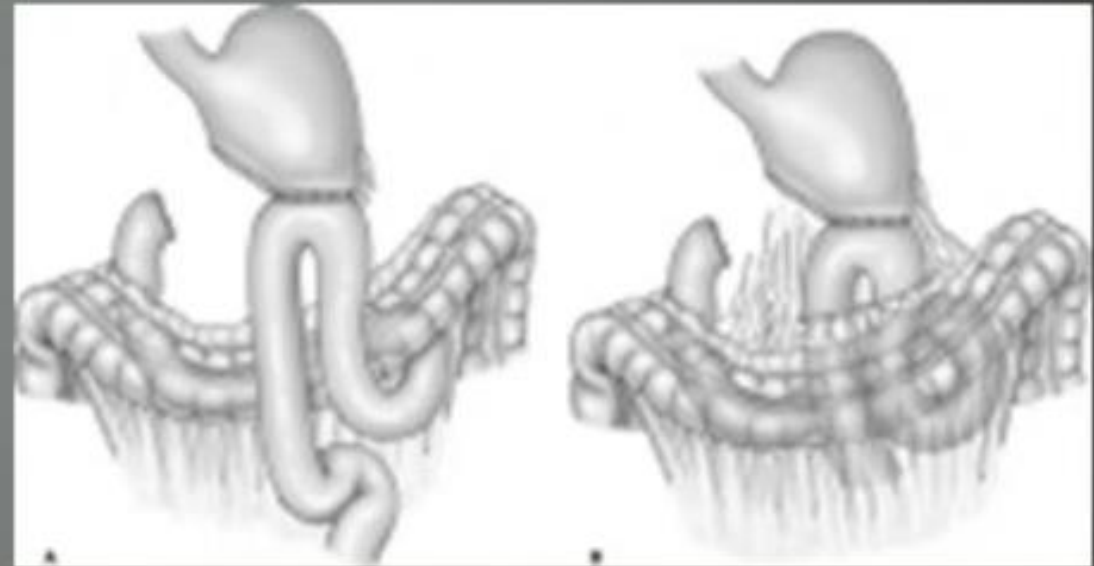




# Billroth II

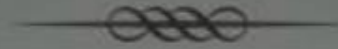


- ❧ Can be used for larger gastric resections
- ❧ Can result in dumping syndrome

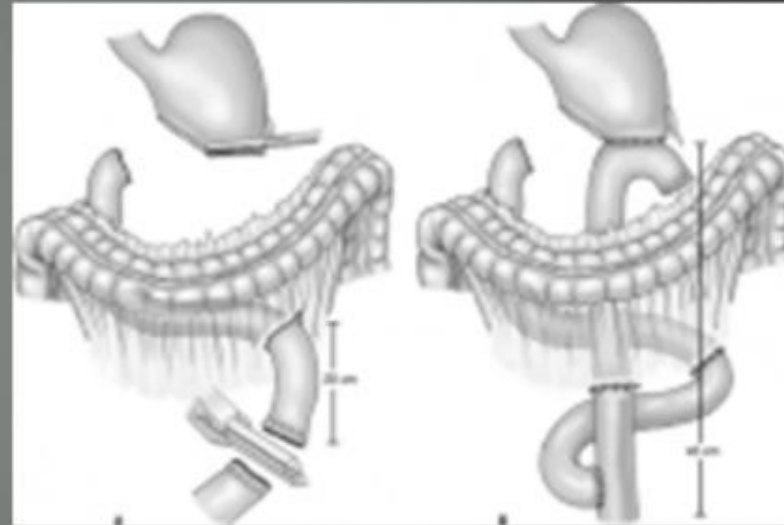




# Roux-en-Y



- ❧ Described by Cesar Roux
  - ❧ Late 1800's
  - ❧ Gastric outlet obstruction
- ❧ Better control of enterogastric reflux
- ❧ Method of choice for early dumping or reflux





# Post-gastrectomy Syndromes

- ▶ A wide spectrum of diseases occurs after gastrectomy
- ▶ Due to denervation of stomach → the pyloric mechanism becomes incompetent and the control of stomach emptying is impaired
- ▶ Early Dumping syndrome
- ▶ Late dumping syndrome
- ▶ Post-vagotomy Diarrhea
- ▶ Afferent loop syndrome / efferent loop syndrome
- ▶ Blind loop syndrome
- ▶ Alkaline Reflux gastritis
- ▶ Roux stasis syndrome
- ▶ Recurrent ulcers
- ▶ Gastric atony
- ▶ Metabolic disturbances



**Table 1.** Post gastrectomy syndromes: classification

Acute complications	<ul style="list-style-type: none"><li>- Anastomotic leak</li><li>- Duodenal stump blowout</li></ul>
Gastric reservoir dysfunction	<ul style="list-style-type: none"><li>- Dumping syndrome (Types I and II)</li><li>- Metabolic alterations</li></ul>
Vagal denervation	<ul style="list-style-type: none"><li>- Gastric stasis and hurry</li><li>- Gallstones</li></ul>
Aberrations of reconstruction	<ul style="list-style-type: none"><li>- Bile reflux gastritis</li><li>- Afferent loop syndrome</li><li>- Efferent loop syndrome</li><li>- Jejunogastric intussusception</li><li>- Roux stasis syndrome</li><li>- Gastro-jejuno-colic fistula</li></ul>
Long term complication	<ul style="list-style-type: none"><li>- Recurrent ulcerations</li><li>- Remnant stump cancer</li></ul>



# Dumping syndrome :

- ▶ Pathophysiology : loss of normal pylorus mechanism , food directly introduced into small bowel
- ▶ **Early dumping :**
  - Occurs 15-30 mins after meal
  - High osmolarity food dumped directly into small bowel → Fluid shifts rapidly from intravascular space into intraluminal space producing acute intravascular volume depletion.
  - tachycardia, diaphoresis, palpitations , Abd pain/cramps, borborygmi , diarrhea, weakness.
  - As simple sugars are absorbed and dilution of hypertonic solution occurs the symptoms decreased.
  - Occurs more with Billroth I, II . LESS in Roux en Y reconstruction.

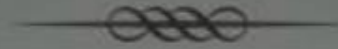


# Late dumping :

- ▶ 2-3 hours after meals, due to excessive insulin release causing transient hypoglycemia and insulin/glucose imbalance .
- ▶ Sudden anxiety, diaphoresis, tremor, tachycardia, palpitations, weakness, fatigue.
- ▶ It is not associated with borborygmi or diarrhea.
- ▶ Pathophysiology:
- ▶ Rapid changes in serum glucose and insulin levels.
- ▶ Large glucose-bolus containing chyme presented to small intestine , glucose is absorbed faster than when the intact pylorus controls emptying of stomach.
- ▶ This causes high levels of serum glucose shortly after meal and causes a big released of insulin. Insulin response exceeds what is necessary to clear glucose from blood and hypoglycemia symptoms happens. Symptoms are due to rapid fluctuations in serum glucose levels.



# Diagnosis



- ❧ Usually made clinically – classic history
  - ❧ Onset of symptoms 10-30mins after meal
  - ❧ Most commonly after high carb meal
  - ❧ Crampy discomfort, belching, nausea, fecal urgency
  - ❧ Light headedness, blurred vision, diaphoresis, flushing, dizziness, hypotension, syncope
  - ❧ Usually no true abdominal pain
- ❧ 50% glucose solution – reproduces symptoms
- ❧ Gastric emptying studies – rapid emptying



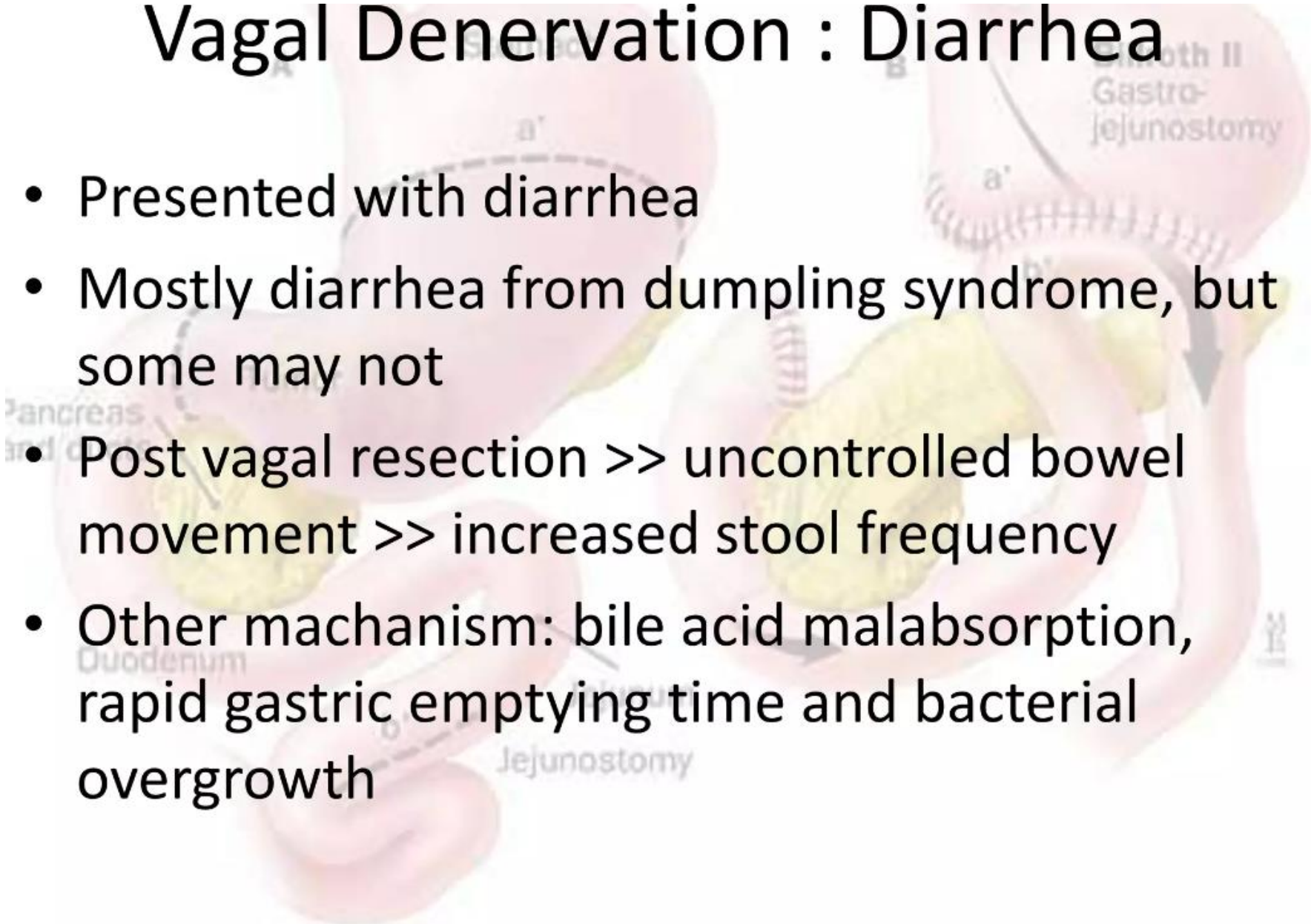
# Treatment of dumping syndrome :

- ▶ Diet modification (most pts will improve)
  - Smaller more frequent meal bulks.
  - Lower carbs in food (less osmolar food)
  - Avoid mixing liquids and solids (Liquids should be ingested before meals or 30 min after meals.
  - Ingest some fat to slow gastric emptying
- ▶ somatostatin analogue (octreotide) for refractory cases.
- ▶ In minority of pts who don't improve → might need conversion of **Billroth** to **Roux en Y** OR creation of anti peristaltic interposition jejunal loop
- ▶ Conversion of **Billroth II** to **Billroth I** can be an option



# Vagal Denervation : Diarrhea

- Presented with diarrhea
- Mostly diarrhea from dumping syndrome, but some may not
- Post vagal resection >> uncontrolled bowel movement >> increased stool frequency
- Other mechanism: bile acid malabsorption, rapid gastric emptying time and bacterial overgrowth



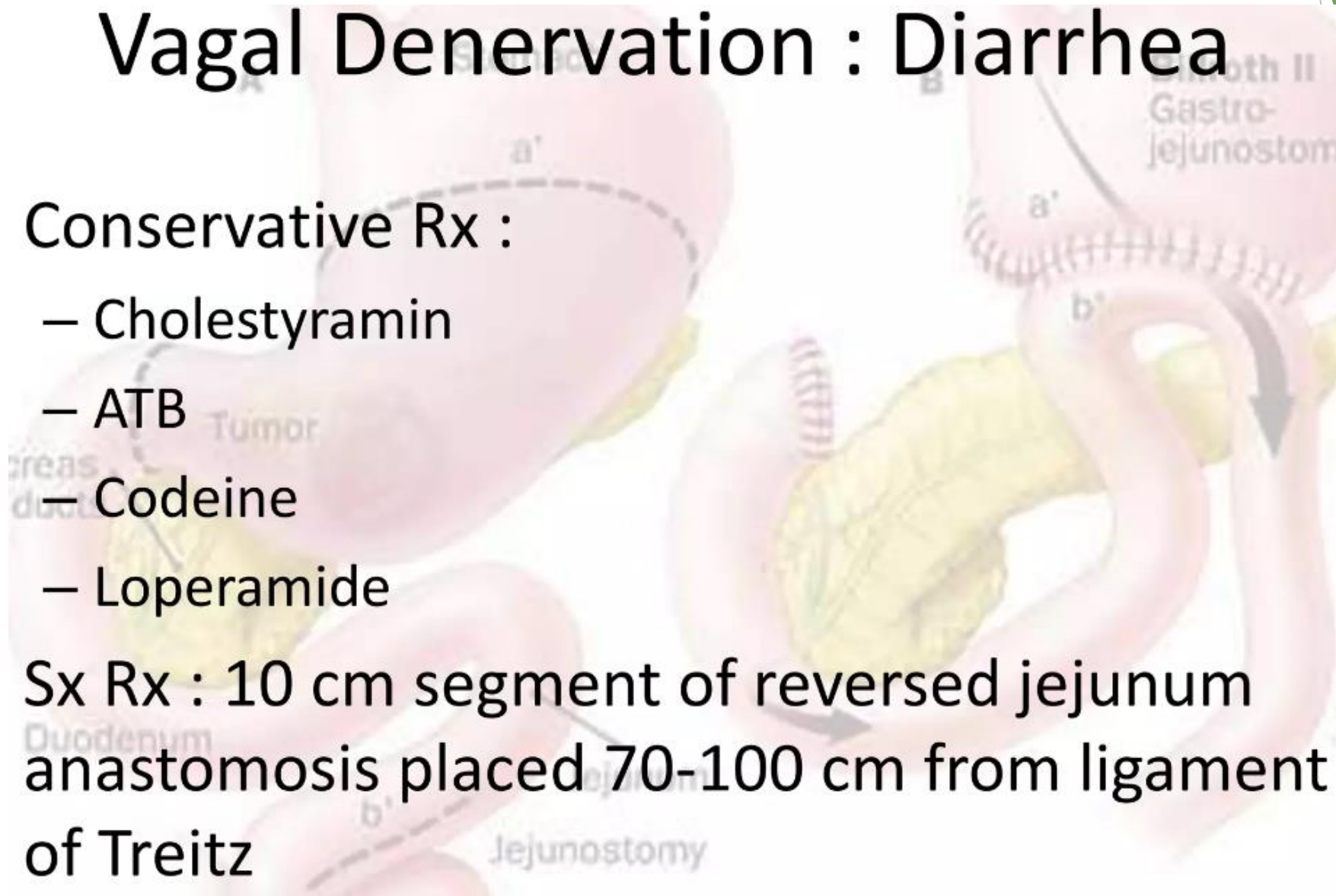


# Vagal Denervation : Diarrhea

Conservative Rx :

- Cholestyramin
- ATB
- Codeine
- Loperamide

Sx Rx : 10 cm segment of reversed jejunum anastomosis placed 70-100 cm from ligament of Treitz





# Vagal Denervation : Gastric stasis

- Conservative Rx :
  - Metoclopramide
  - Domperidone
  - Erythromycin
- NJ tube feed





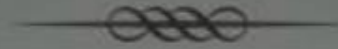
# Gastroparesis



- ❧ Most common syndrome
- ❧ ~50% of pts thought to have symptoms
- ❧ Definition is highly variable
  - ❧ Hard to get true incidence



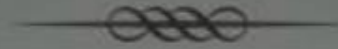
# Diagnosis



- ❧ Most pts diagnosed if not taking adequate po intake 7-14 days post-op after gastric procedure
- ❧ Symptoms: nausea, bloating, fullness, early satiety, vomiting
- ❧ Gastric emptying studies – normal: 60% solid, 80% liquid clearance at 60 min.
  - ❧ Nuclear medicine or thin barium vs gastrograffin



# Acute Gastroparesis



## Causes:

### ❧ Metabolic/Neuronal

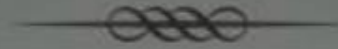
- ❧ Release of norepi and inhibition of acetylcholine in immediate post-op period
- ❧ Electrolytes – hypomagnesemia, hypokalemia
- ❧ Endocrine – hypothyroidism, DM
- ❧ Medications – opiates, anticholinergics, antidepressants

### ❧ Functional

- ❧ Preoperative gastric outlet obstruction-affects contraction
- ❧ Effects of truncal vagotomy – mostly solids
- ❧ Stomal edema, adhesions, kinking, hematoma, intussusception



# Treatment



## Conservative

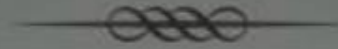
- ❧ NGT decompression
- ❧ Prokinetic agents
  - ❧ Bethanechol, Reglan, Erythromycin Cisapride – off market
- ❧ Correction of lytes
- ❧ patience

## After failed treatment

- ❧ Minimum of 3-4 wks
  - ❧ No improvement – re-explore
  - ❧ Look for mechanical causes
  - ❧ Place feeding tube – jejunostomy
- ❧ Gastric pacing – experimental



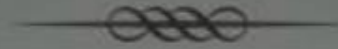
# Chronic Gastroparesis



- ❧ Diagnosis of exclusion – rule out stricture, internal hernia, stomal edema, intussusception
- ❧ ~2% of patients after gastric surgery – mostly after truncal vagotomy
- ❧ Symptoms start later in the post-op period



# Chronic Gastroparesis

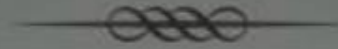


## Diagnosis

- ❧ Symptoms – early satiety, nausea, vomiting, postprandial bloating, hiccups, belching
  - ❧ Increase throughout the day
- ❧ Emesis of food ingested days earlier – pathognomonic
- ❧ Need UGI and EGD to rule out other syndromes



# Treatment



## Conservative treatment

- ✎ Same as acute gastroparesis
- ✎ More emphasis on maximizing response to prokinetic agents

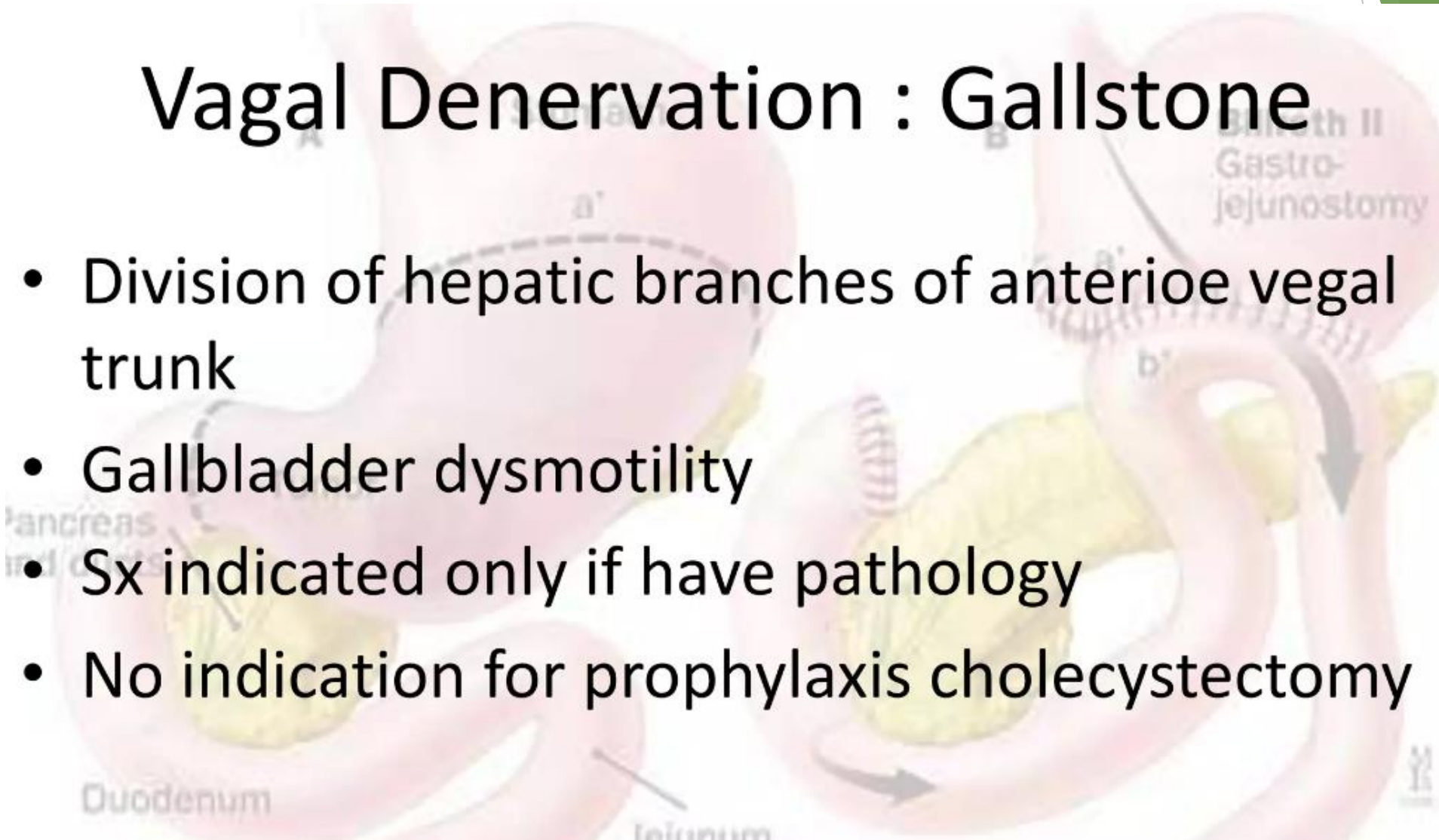
## Surgical treatment

- ✎ Resection of atonic portion
- ✎ Using a different type of reconstruction
- ✎ Only total gastrectomy may be curative
- ✎ Gastric pacing – some improvement at 6 mo, but disappears at 1 yr



# Vagal Denervation : Gallstone

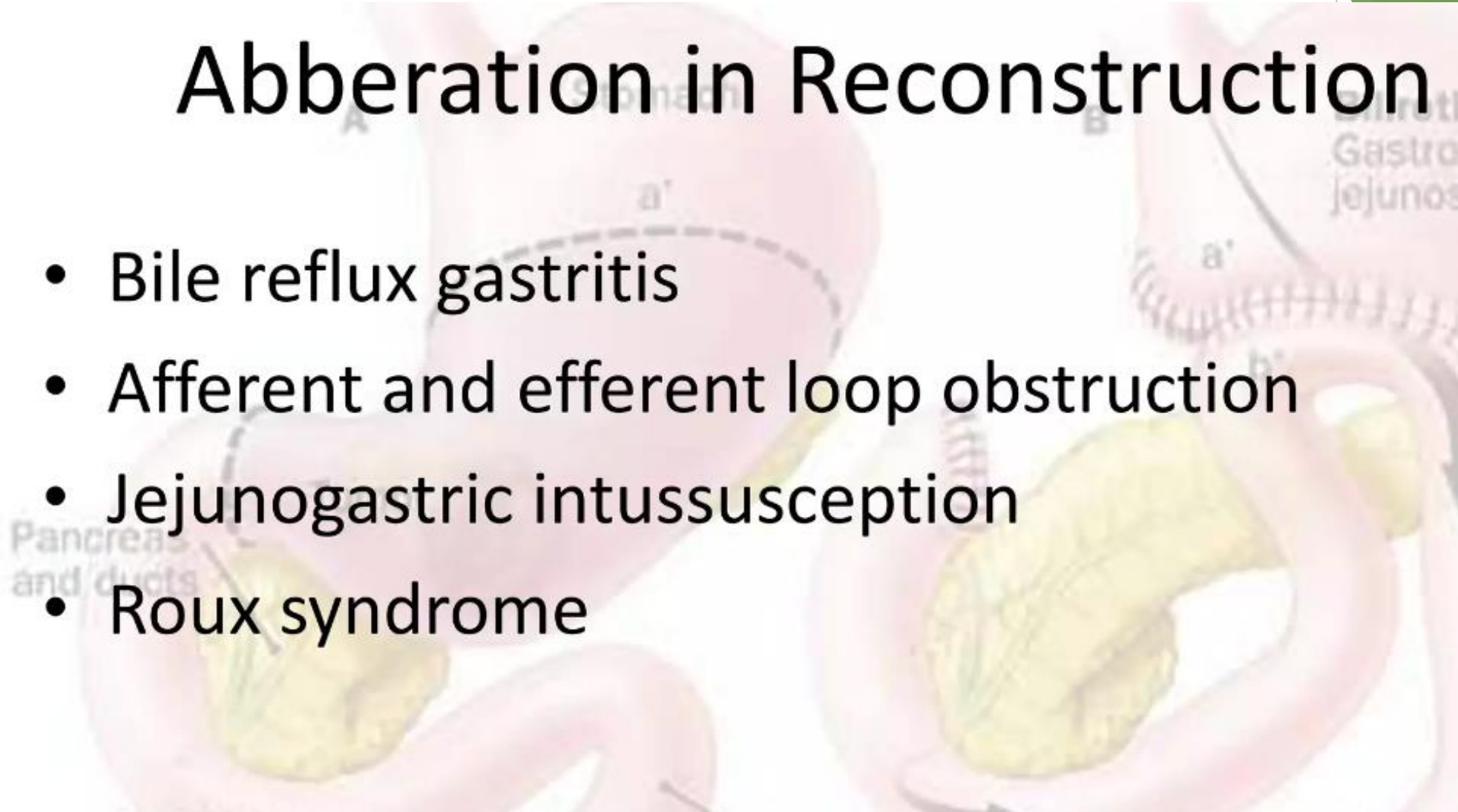
- Division of hepatic branches of anterior vagal trunk
- Gallbladder dysmotility
- Sx indicated only if have pathology
- No indication for prophylaxis cholecystectomy





# Abberation in Reconstruction

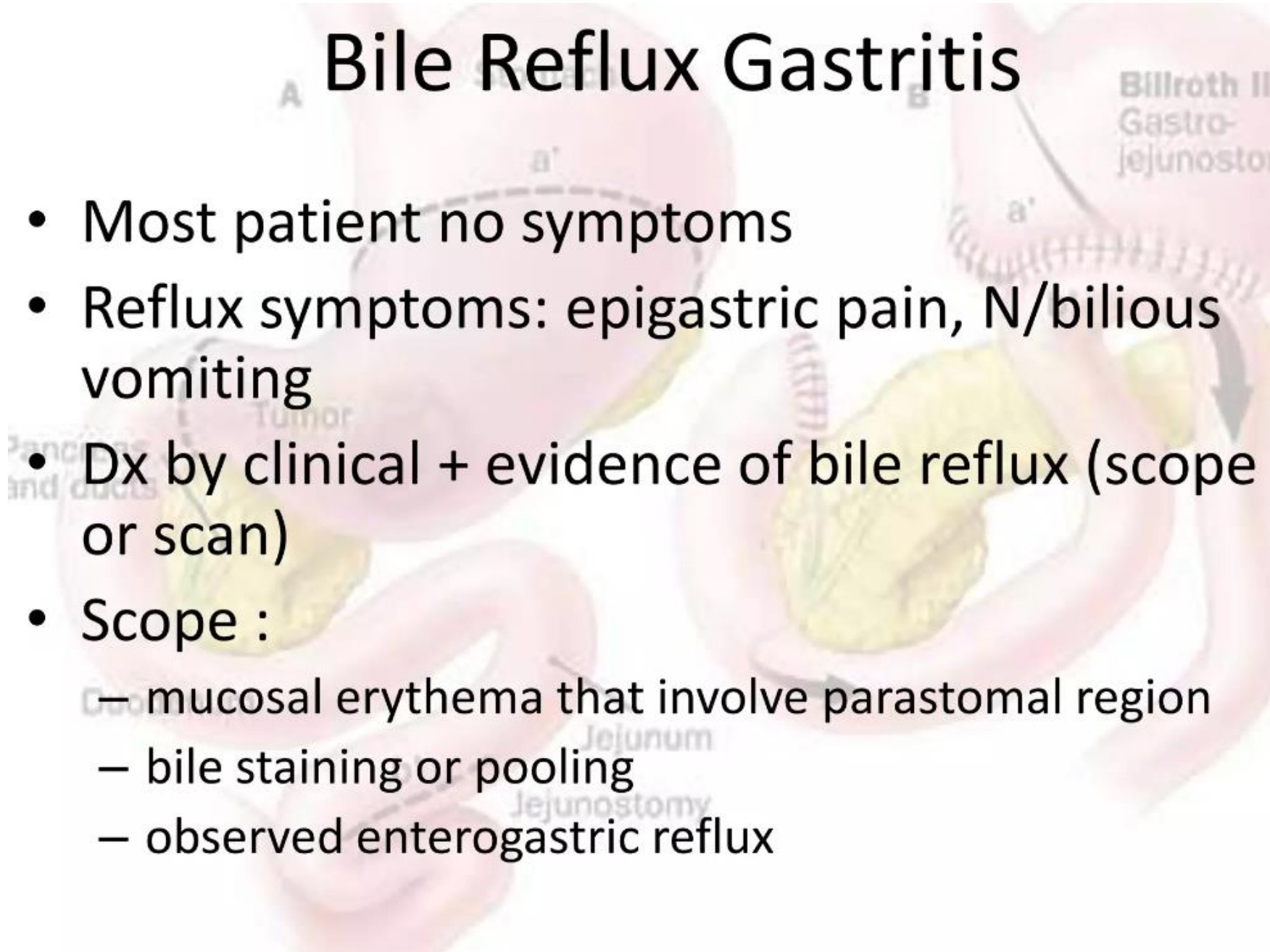
- Bile reflux gastritis
- Afferent and efferent loop obstruction
- Jejunogastric intussusception
- Roux syndrome





# Bile Reflux Gastritis

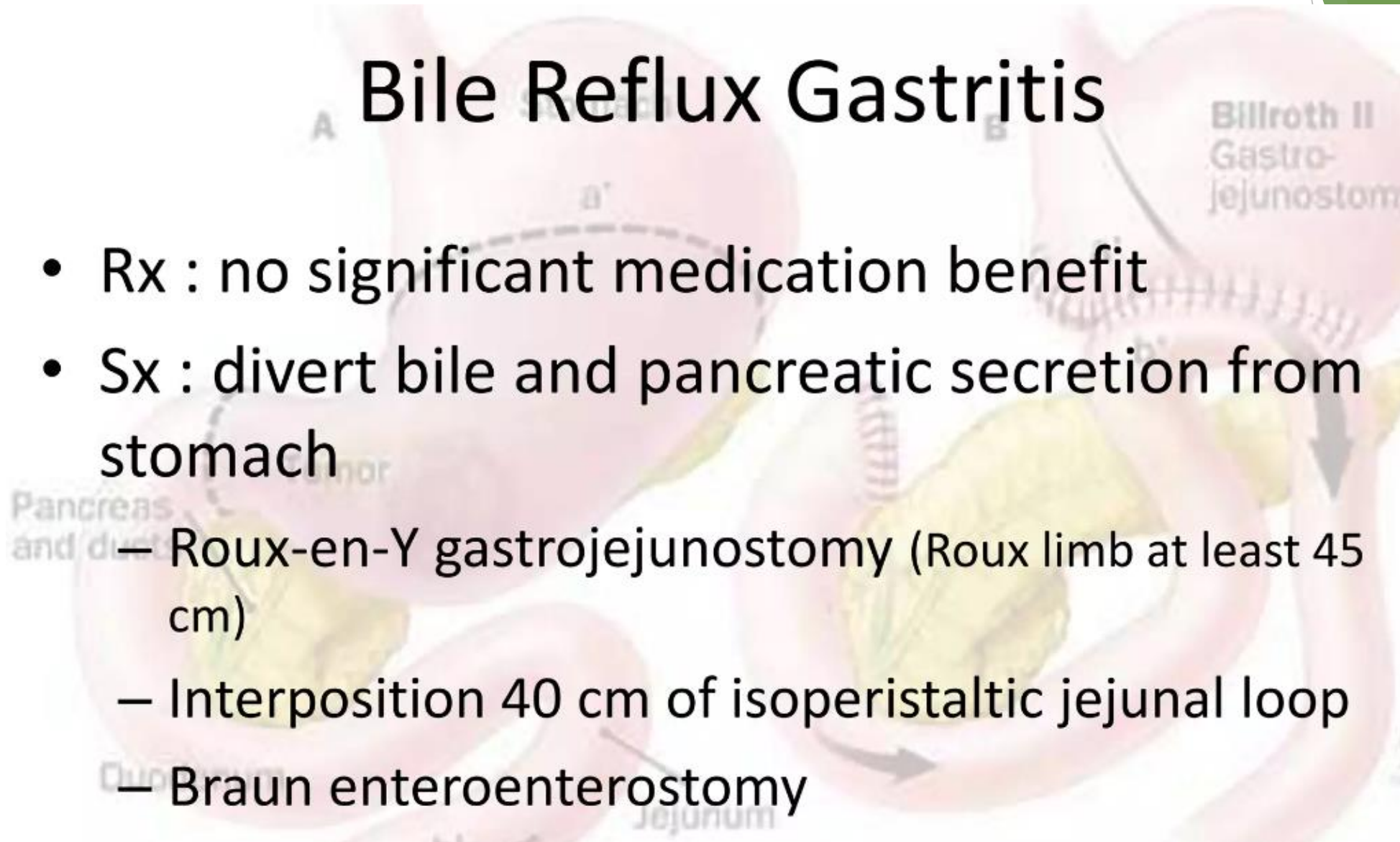
- Most patient no symptoms
- Reflux symptoms: epigastric pain, N/bilious vomiting
- Dx by clinical + evidence of bile reflux (scope or scan)
- Scope :
  - mucosal erythema that involve parastomal region
  - bile staining or pooling
  - observed enterogastric reflux





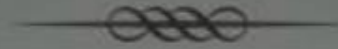
# Bile Reflux Gastritis

- Rx : no significant medication benefit
- Sx : divert bile and pancreatic secretion from stomach
  - Roux-en-Y gastrojejunostomy (Roux limb at least 45 cm)
  - Interposition 40 cm of isoperistaltic jejunal loop
  - Braun enteroenterostomy





# Alkaline Reflux Gastritis



- ❧ Reflux of intestinal contents into the stomach
  - ❧ Significant mucosal injury causing symptoms
- ❧ Debilitating in 1-2% of post gastrectomy pts.
- ❧ ~20% of healthy controls have duodenogastric reflux
  - ❧ Also increased in pts with cholelithiasis/cholecystectomy
- ❧ More common in Billroth II > B I > V&P



# Pathophysiology



- ❧ Refluxate contents – bile, pancreatic enzymes, other intestinal secretions
- ❧ Some disagreement – bile salts + gastric acid = decreased barrier function of mucosa
  - ❧ Causes gastritis and symptoms



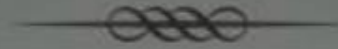
# Diagnosis



- ❧ Again a diagnosis of exclusion
- ❧ Symptoms: usually start ~1yr post-op
  - ❧ Fatigue, malaise, weight loss
  - ❧ Constant, burning epigastric pain, worse with food
  - ❧ Persistent nausea, always bilious emesis
    - ❧ Pain characteristically not alleviated with emesis



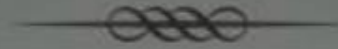
# Diagnosis



- ❧ EGD – classic beefy red, edematous gastric mucosa
  - ❧ Sharp demarcation of mucosa at anastomosis
- ❧ HIDA – to evaluate gastric reflux index
  - ❧ <5% - healthy, normal
  - ❧ 5% - 45% - asymptomatic, after BII
  - ❧ >60% - symptomatic
- ❧ Bernstein test – reproduction of symptoms with gastric infusion of Na hydroxide



# Treatment



## Conservative

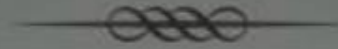
- ❧ Doesn't work as well
- ❧ High fat/protein diet
- ❧ PPI, carafate, reglan
- ❧ Cholestyramine – binds bile
- ❧ Most promising –  
Ursodeoxycholic acid  
Resolution of symptoms

## Surgical

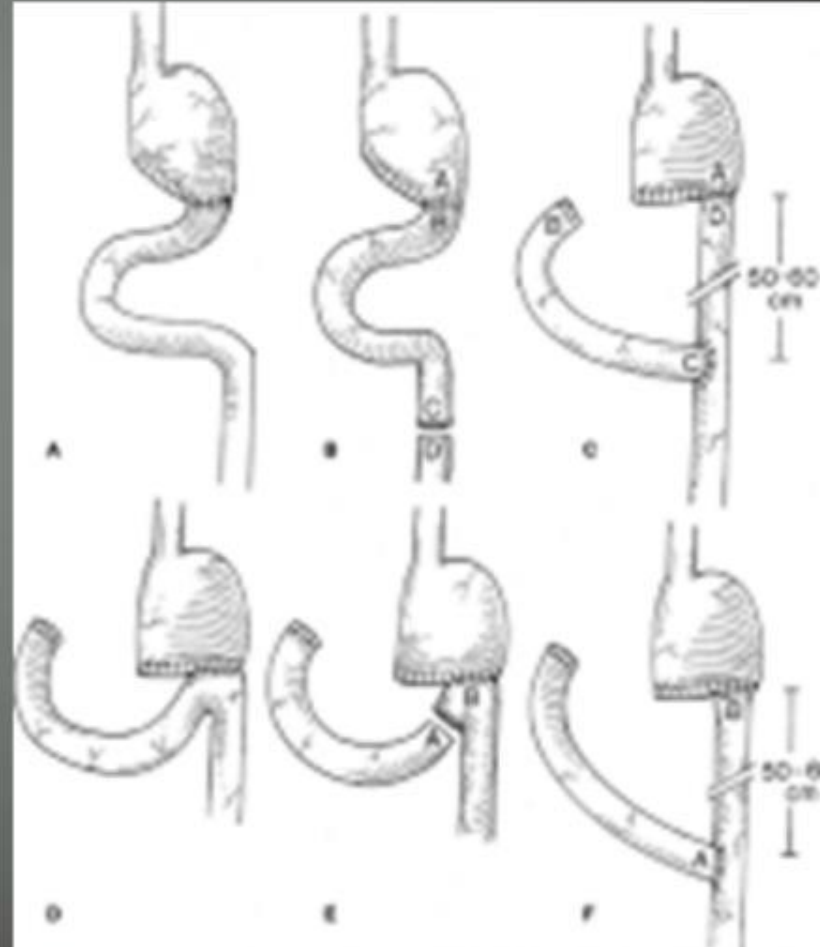
- ❧ Braun procedure
- ❧ Distal enteroenterostomy
- ❧ Henley loop
- ❧ Roux-en-Y reconstruction



# Roux-en-Y Reconstruction



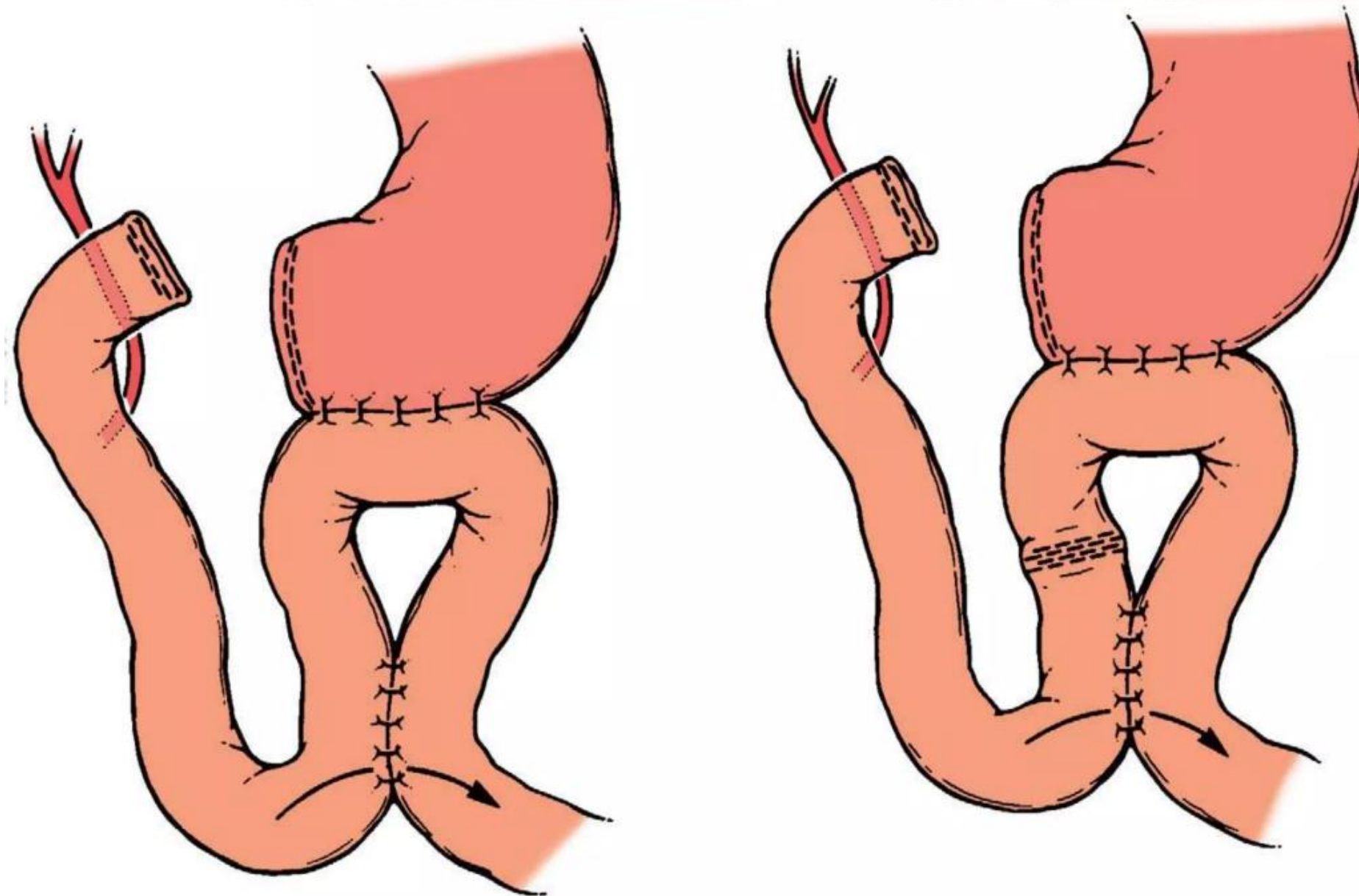
- ❧ Procedure of choice
- ❧ Long Roux limb – 45-60cm
- ❧ Symptoms relieved in 80% of pts
- ❧ Ulcerogenic, ~30% gastroparesis
  - ❧ Evaluate original path for complete antrectomy and gastric motility





# Bile Reflux Gastritis

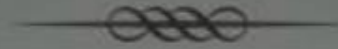
Billroth II



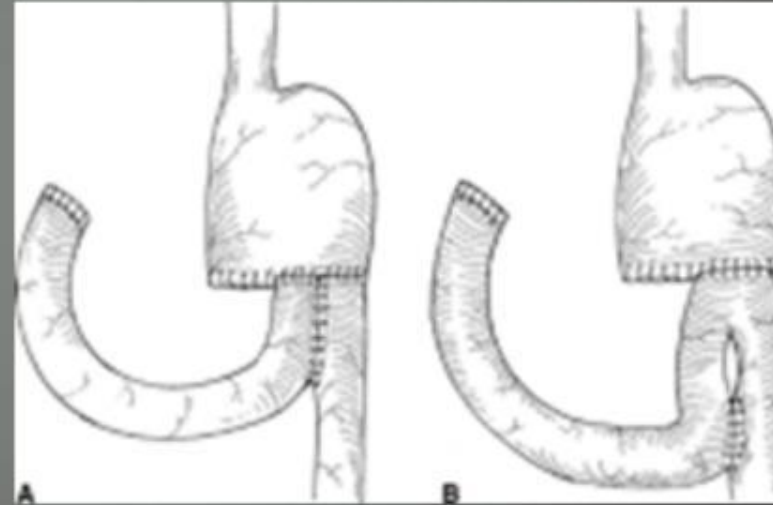


# Braun Procedure

## Distal enteroenterostomy



- Both decrease amount of refluxant but not enough protection from reflux
- Goal should be to divert all intestinal contents away from gastric remnant





# Afferent and Efferent Limb Syndromes



- ❧ Caused by partial or complete obstruction of jejunal limbs
- ❧ Characteristic signs and symptoms
- ❧ Afferent is more common than efferent

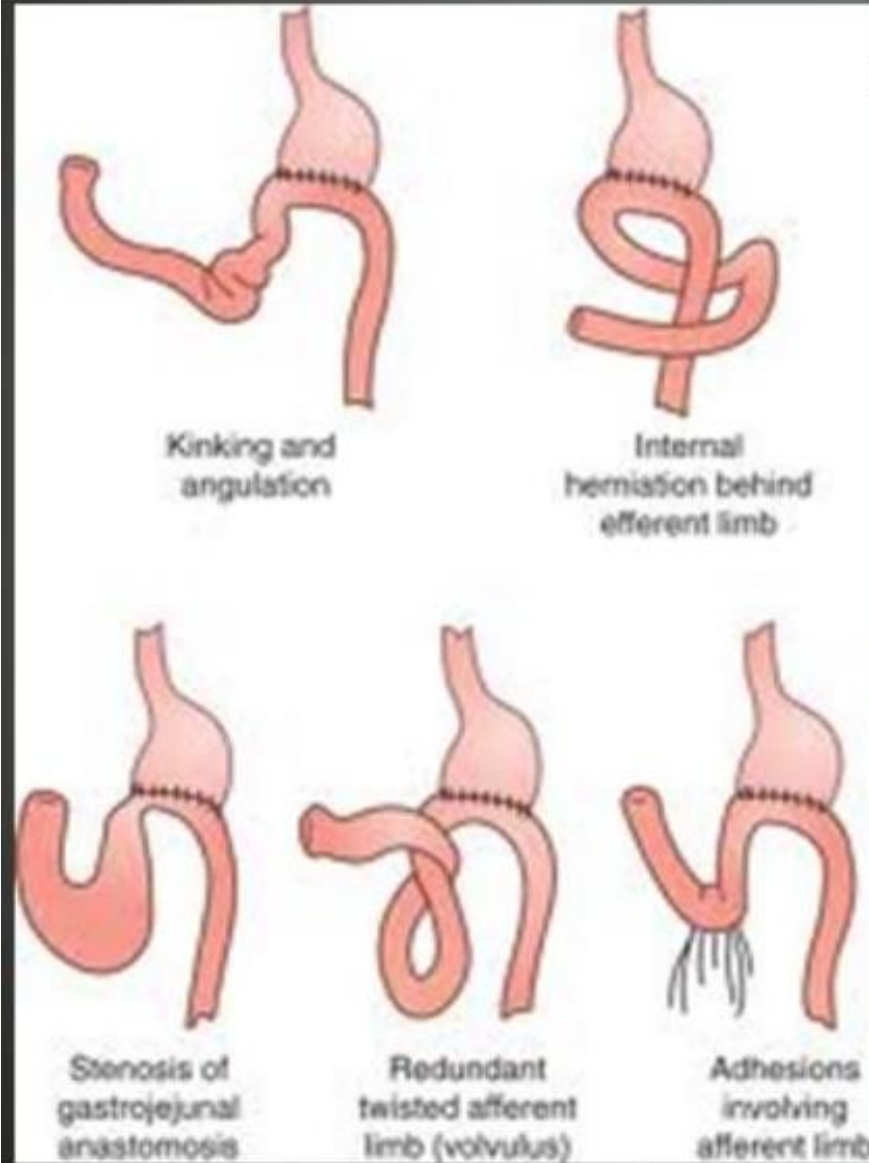


# Afferent and Efferent Loop Obstruction

- Afferent loop syndrome
  - Afferent limb length > 30-40 cm can be obstruction
  - Chronic > acute
  - Severe postprandial epigastric pain(30-60 mins)
  - Projectile bilious vomiting
  - Dramatic clinical relief after vomiting
  - Some can be presented with diarrhea



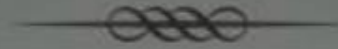
# Afferent Limb Syndrome



- Occurs only with BII
- Almost always due to too long of a limb
- Acute and chronic forms
- Acute = closed loop obstruction, usually immediately post-op, needs surgery



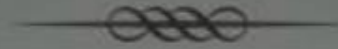
# Diagnosis



- ❧ Symptoms of intermittent RUQ pain, relieved with vomiting
- ❧ Hyperamylasemia
  - ❧ ?pancreatitis
- ❧ EGD, gastrograffin study, CT – dilated loop
- ❧ HIDA – delayed films can show +/- obstruction



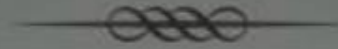
# Chronic Afferent Limb Syndrome



- ❧ More common than acute
- ❧ Due to partial limb obstruction
- ❧ Increased incidence if anastomosis is retrocolic and above the mesocolonic defect
- ❧ Bacterial overgrowth – blind loop syndrome, vit B12 deficiency



# Manifestations of Chronic Afferent Limb Syndrome and Alkaline Reflux Gastritis



## Chronic Afferent Limb Syndrome

- ❧ Pain after meals
- ❧ Vomitus: Bile, no food, relieves pain, projectile
- ❧ Occult bleeding rare
- ❧ Cause: limb obstruction

## Alkaline Reflux Gastritis

- ❧ Pain unrelated to meals
- ❧ Vomitus: Bile and food, no change in pain, nonprojectile
- ❧ Occult bleeding common
- ❧ Cause: enterogastric reflux

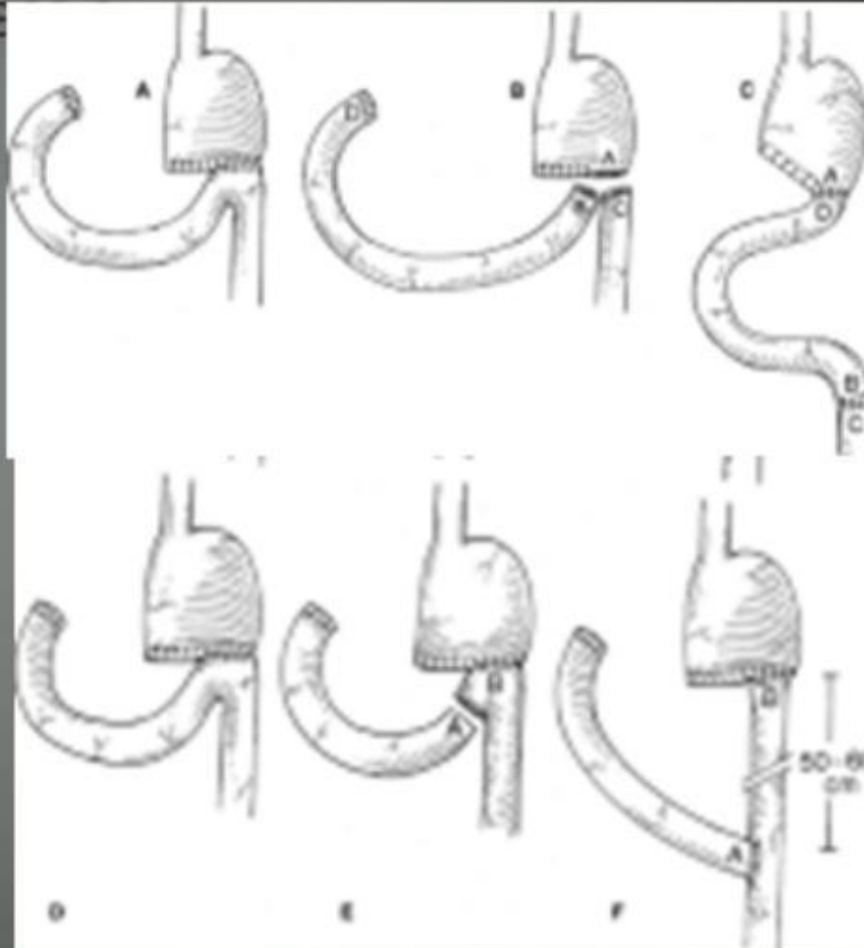


# Treatment

Surgical

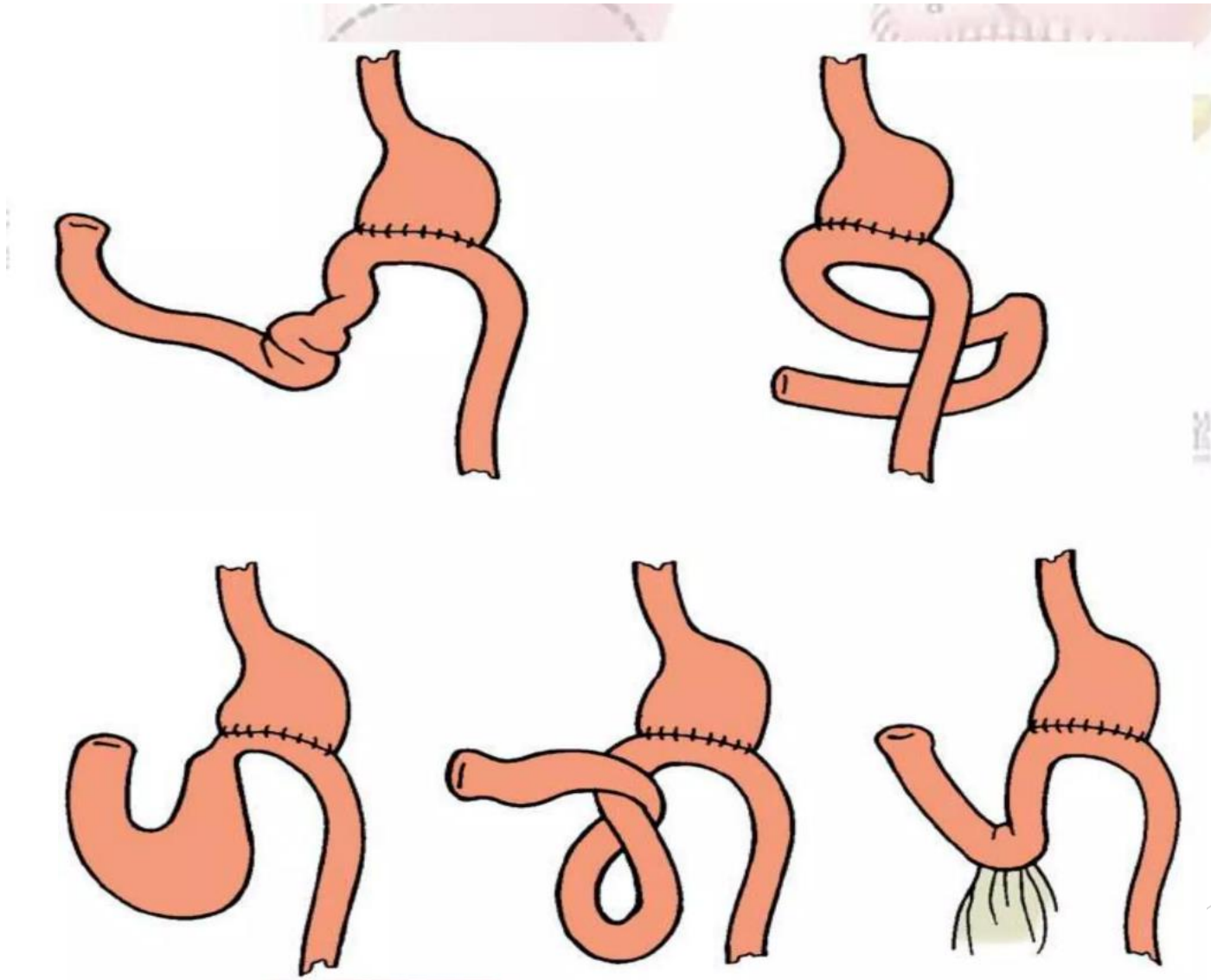
Two most accepted

- ❧ Convert BII to BI
  - ❧ Can be difficult to get to duodenal stump
- ❧ Roux-en-Y – important to remember to perform complete vagotomy
  - Prevents marginal ulcers



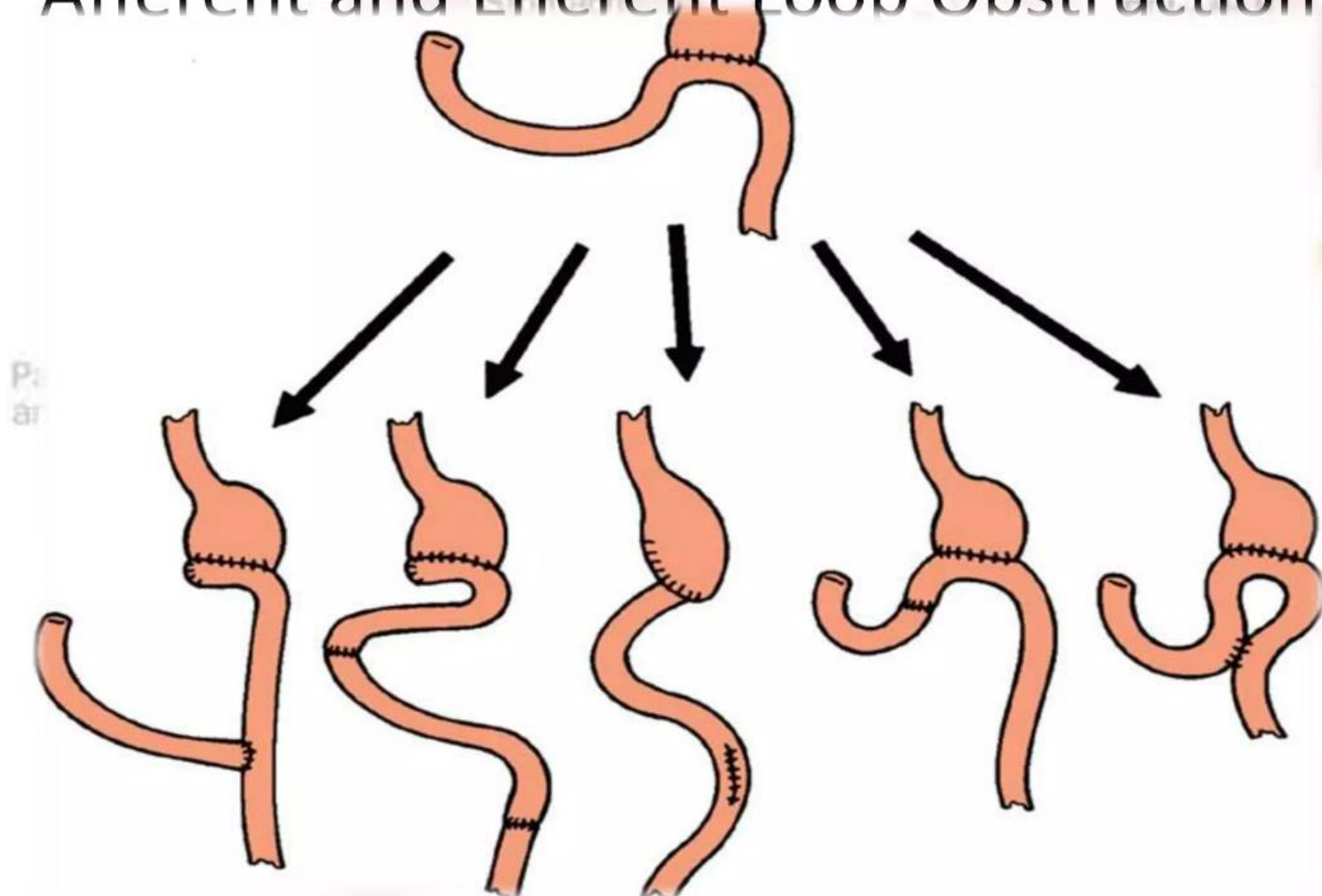


# Afferent loop syndrome

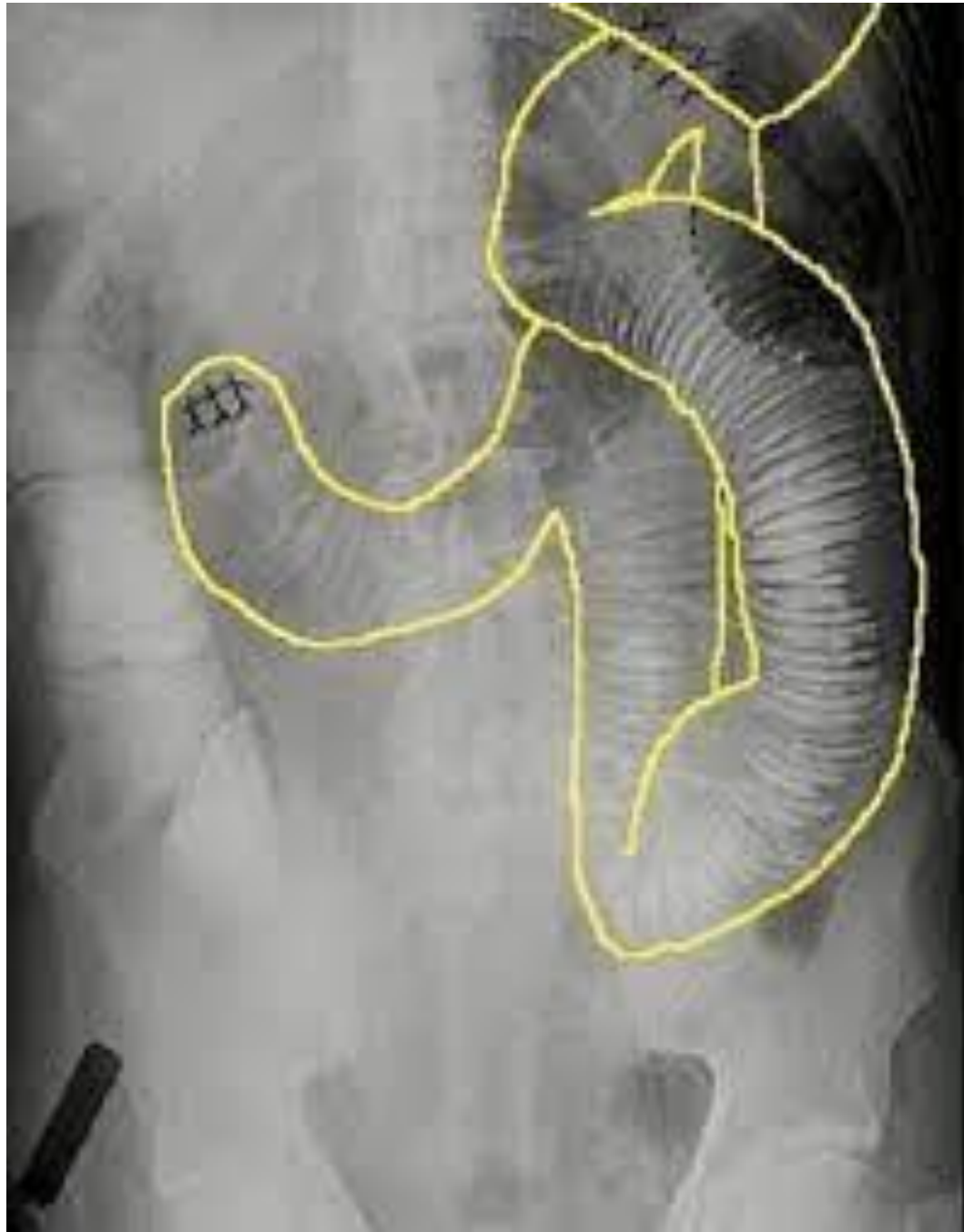




# Afferent and Efferent Loop Obstruction

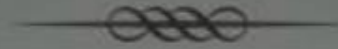








# Efferent Limb Syndrome

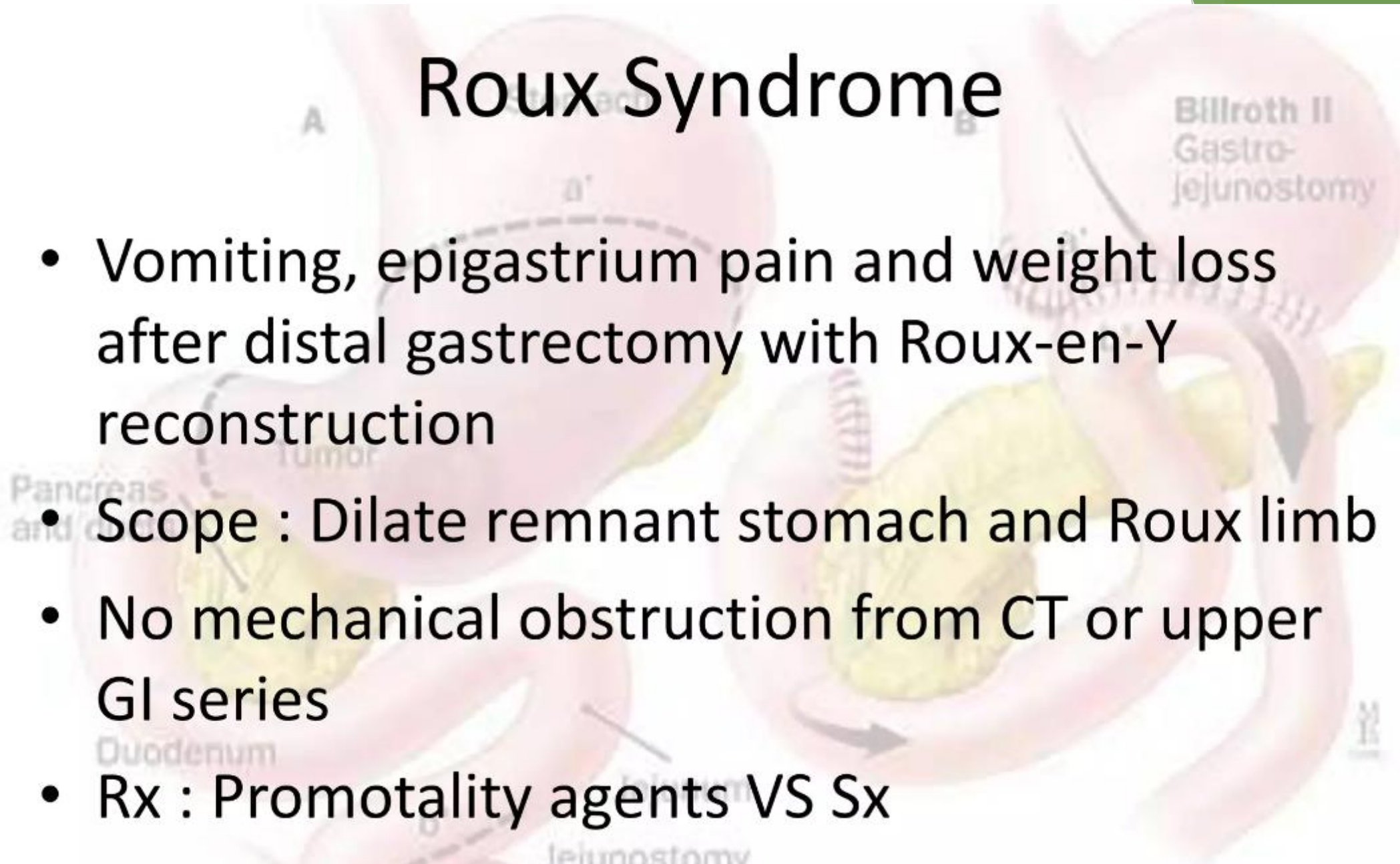


- ❧ Less common and harder to diagnose
- ❧ Symptoms of crampy LUQ and epigastric pain associated with bilious vomiting
- ❧ Most commonly caused by internal herniation of the limb behind the anastomosis
- ❧ Diagnosed with barium UGI and EGD
- ❧ Treatment is surgical and is determined at the time of exploration



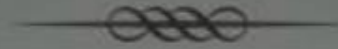
# Roux Syndrome

- Vomiting, epigastrium pain and weight loss after distal gastrectomy with Roux-en-Y reconstruction
- Scope : Dilate remnant stomach and Roux limb
- No mechanical obstruction from CT or upper GI series
- Rx : Protonality agents VS Sx





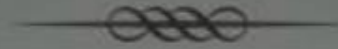
# Roux Limb Syndrome



- ❧ 25-30% incidence of nausea, vomiting, postprandial epigastric fullness, and upper abdominal pain
- ❧ Hard to differentiate from gastroparesis
  - ❧ Usually a late complication
- ❧ True cause is unknown
  - ❧ Thought to occur due to disrupted jejunal myoelectric propagation
  - ❧ Loss of vagal stimulation that accompanies gastrectomy
  - ❧ No true correlation between length of Roux limb



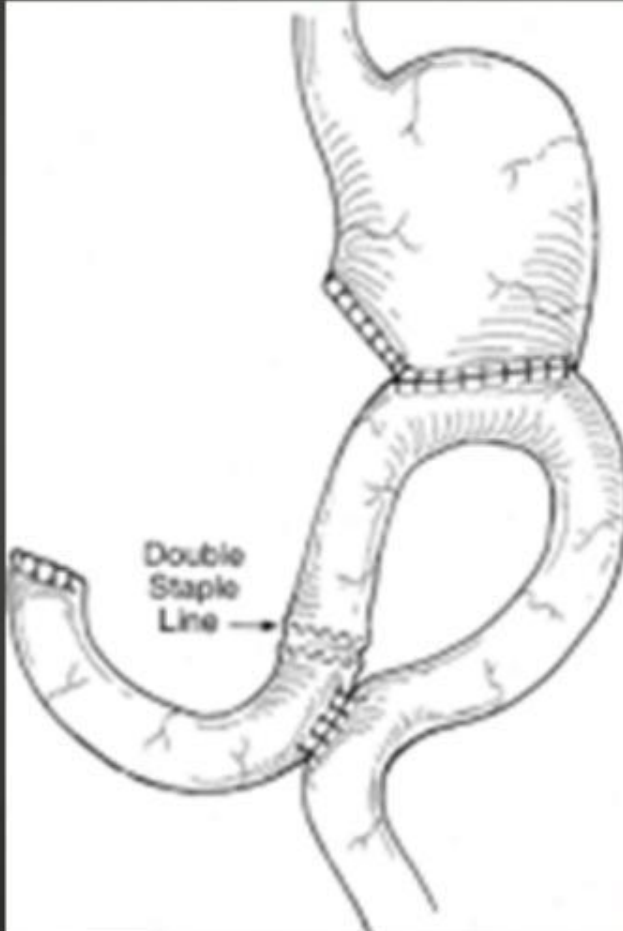
# Diagnosis



- ❧ Symptoms months to years after gastrectomy
- ❧ UGI and EGD usually normal
- ❧ Gastric emptying – significantly delayed
- ❧ Symptoms are not improved with prokinetic agents
  - ❧ Main way to differentiate from gastroparesis
- ❧ Don't improve over time



# Treatment



- ❧ Surgical
- ❧ Further gastric resection with new Roux-en-Y
  - ❧ To remove atonic stomach
- ❧ Conversion to BII with Braun 30cm distal
- ❧ “Uncut Roux”
  - ❧ Avoids defunctionalized intestine



## Late dumping

**Clin presentation is the** same as early dumping but happens hours after eating.

**Mechanism:** Secondary to hypoglycemia from a postprandial insulin peak.

**Treatment:** Dietary changes (frequent small meals that are high in protein and fat and low in carbohydrates for early dumping; increased carbohydrate consumption for late dumping).  
If intractable (rare)-(Conversion of a vagotomy and pyloroplasty to an antrectomy with Roux-en-Y reconstruction)

## Early Dumping

Occurs in 20 % of patients after gastrectomy or vagotomy and drainage.

**Presentation** is with postprandial GI discomfort, which may include N+V+D and cramps, with vasomotor symptoms eg. diaphoresis, palpitations, and flushing.

**Mechanism involves** a rapid emptying of hyperosmolar chyme (particularly carbohydrate) into small bowel. Leads to net fluid retention due to the osmotic gradient + releases one or more vasoactive hormones, eg. serotonin and VIP

## Alkaline reflux gastritis

Occurs in 2% post-op

**Clin Presentation involves** persistent burning epigastric pain and chronic nausea that is aggravated by meals.

**Diagnosis** is usually diagnosis of exclusion - OGD may show gastritis/ technetium biliary scan can demonstrate increased reflux of bile into the stomach.

**There are no effective treatments.**

## Miscellaneous —

Weight Loss and Dietary Restriction (mainly to reduce symptoms of eg early satiety)

Malabsorption - accelerated gastric emptying/ reduced gastric seiving (Particles >1mm go through)/ decreased pancreatic function

Bacterial overgrowth

Iron/( 50%)/ B12(20%)/Folate( 40%)

Metabolic bone disease

## Early satiety —

**It is caused by** postsurgical atony, gastric stasis as a result of denervation, or from the "small gastric remnant syndrome" related to resection.

**Clin Presentations involves** epigastric fullness with meals, often followed by emesis.

**Diagnosis and**

Atony can be identified with a solid food emptying test and may respond to prokinetic agents such as metoclopramide and erythromycin. If these fail, completion gastrectomy may be required, although there is some anecdotal evidence that gastric pacing may prove useful.

The "small gastric remnant syndrome"- small and frequent meals

## Afferent and efferent loop syndromes

Post Billroth II reconstruction or gastroenterostomy.

**It is due to** mechanical obstruction of the limbs by kinking, anastomotic narrowing, or adhesions.

**Clin Presentation involves** postprandial epigastric pain and nonbilious vomiting relieved by projectile bilious vomiting. The detection of distended afferent loop on CT is diagnostic.

**Treatments include** conversion to a Roux-en-Y anastomosis.

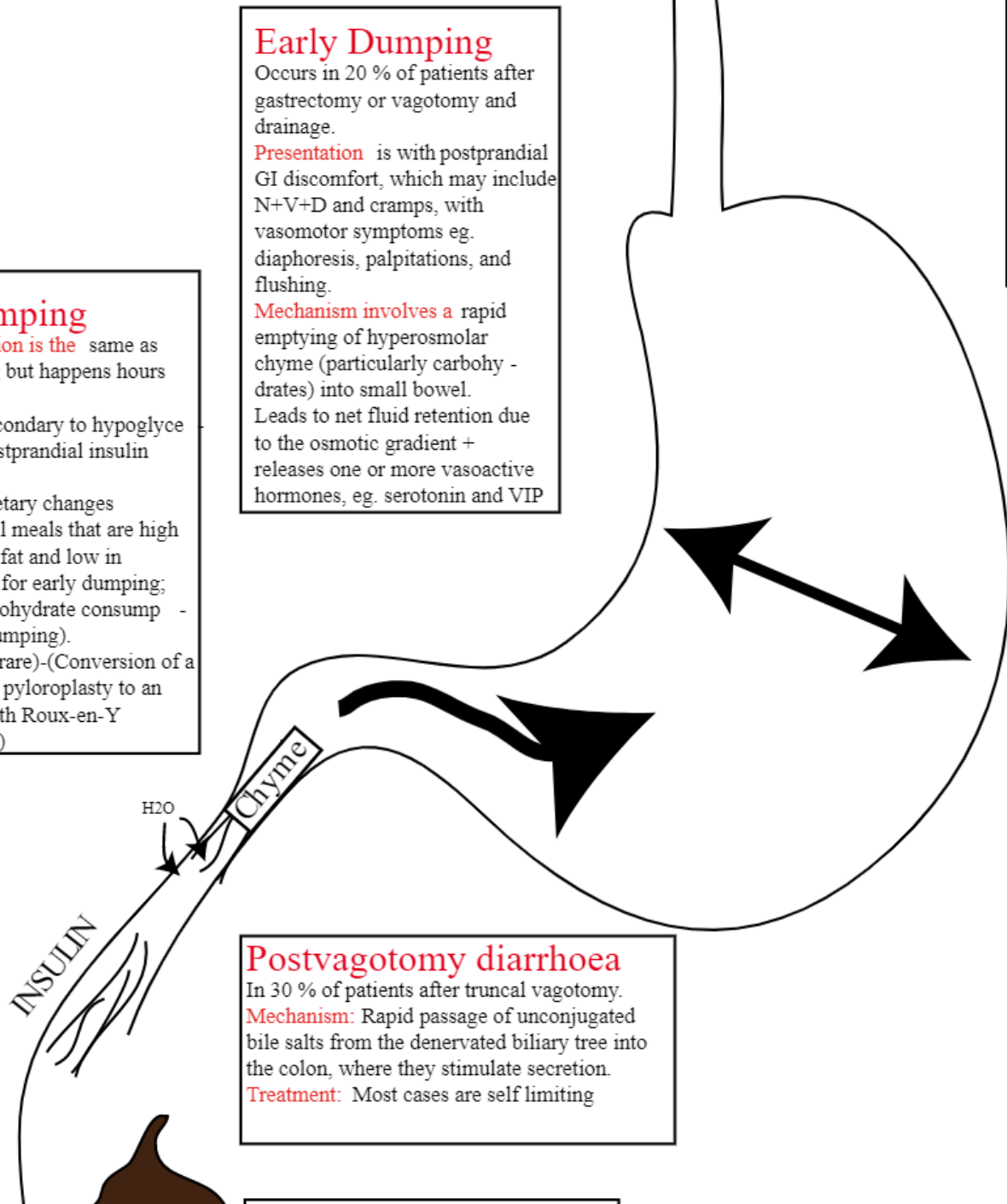
## Afferent and efferent loop syndromes —

## Postvagotomy diarrhoea

In 30 % of patients after truncal vagotomy.

**Mechanism:** Rapid passage of unconjugated bile salts from the denervated biliary tree into the colon, where they stimulate secretion.

**Treatment:** Most cases are self limiting





# TREATMENT SUMMARY

Dumping syndrome	<ul style="list-style-type: none"><li>• Interposition of jejunal loop: Iso/anti-peristaltic.</li><li>• Conversion of loop gastrojejunostomy to RouxenY reconstruction (57,58).</li></ul>
Post vagotomy diarrhoea	<ul style="list-style-type: none"><li>• Anti-peristaltic jejunal loop distal interposition (59).</li></ul>
Gastric stasis	<ul style="list-style-type: none"><li>• Gastric pacing/Electric pacemaker (60).</li><li>• Venting gastrostomy.</li><li>• Re-operative near total gastrectomy.</li></ul>
Bile reflux gastritis	<ul style="list-style-type: none"><li>• Conversion to a Roux-en-Y reconstruction.</li></ul>
Afferent loop syndrome and Efferent loop syndrome	<ul style="list-style-type: none"><li>• Endoscopic insertion of double pigtail stent (61).</li><li>• Conversion to RouxenY.</li><li>• Conversion to Billroth I.</li><li>• Braun entero-enterostomy.</li></ul>
Jejuno-gastric intussusception	<ul style="list-style-type: none"><li>• Revision of the GJ/Resection of the intussuscepted segment.</li><li>• Revision of the GJ/Resection of the Conversion to a Rouxen Y.</li></ul>
Roux stasis syndrome	<ul style="list-style-type: none"><li>• Resection followed by RouxenY revision.</li><li>• Avoid Roux reconstruction in &gt;50% resection of stomach (BII with Braun entero-enterostomy preferred) (62).</li></ul>



# Jejunal Interposition

- ❧ Isoperistaltic
  - ❧ 10-20cm
  - ❧ Regulate rate of gastric emptying and dilate to inc reservoir
- ❧ Antiperistaltic
  - ❧ No longer than 10cm
  - ❧ Slow gastric emptying
  - ❧ Gastric retention, reflux, obstructive symptoms





# Gastric Cancers (GC)



# Gastric Cancers (GC)

## Common Primary

- Adenocarcinoma (95%),
- Lymphoma (4%),
- Gastrointestinal stromal tumors GIST (1%)

## Rare Primary

- Carcinoid, Angiosarcoma, Carcinosarcoma, and Squamous cell carcinoma

## Secondary From :

- Melanoma , Breast(Blood born)
- Colon or Pancreas (Direct ext.)
- Ovary (By peritoneal seeding )

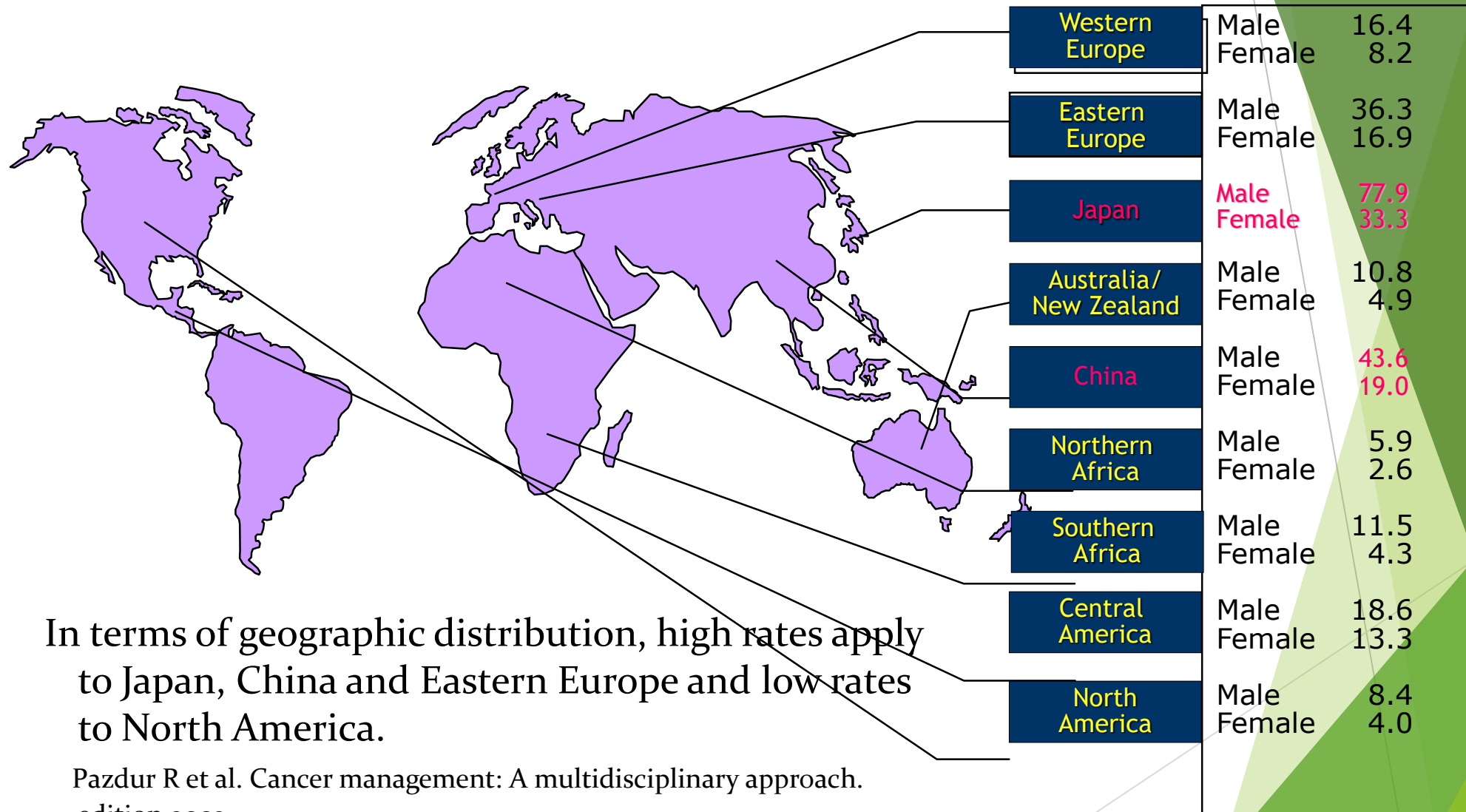


# Gastric Adenocarcinoma:

- ▶ Worldwide : Fifth most common cancer and third most common cause of cancer related deaths.
- ▶ Incidence and mortality are declining in developed countries.
- ▶ Twice higher incidence and mortality in blacks/Asians.
- ▶ Male to female 2:1
- ▶ Geographical discrepancy (higher incidence in Japan/East Asia).
- ▶ Requires treatment in a high volume tertiary centers
- ▶ Multidisciplinary approach for treatment offers better outcome (nutritional,endoscopic,surgical,medical, radiation oncologists)



# GC Worldwide incidence





# Risk factors :

## ► *H.pylori* infection :

- Usually with prolonged infection >10 years
- Distal gastric ca, intestinal type in 90%
- Atrophic gastritis → metaplasia → dysplasia → cancer

## ► Diet :

- salted, smoked, poorly preserved food
- Nitrate food (formation of N-Nitroso carcinogen)
- Lower risk with fruits, vegetables, vit C, vit A, antioxidants.

## ► Occupations: metal, mines, rubber, wood, asbestos workers

## ► Obesity



# Risk factors :

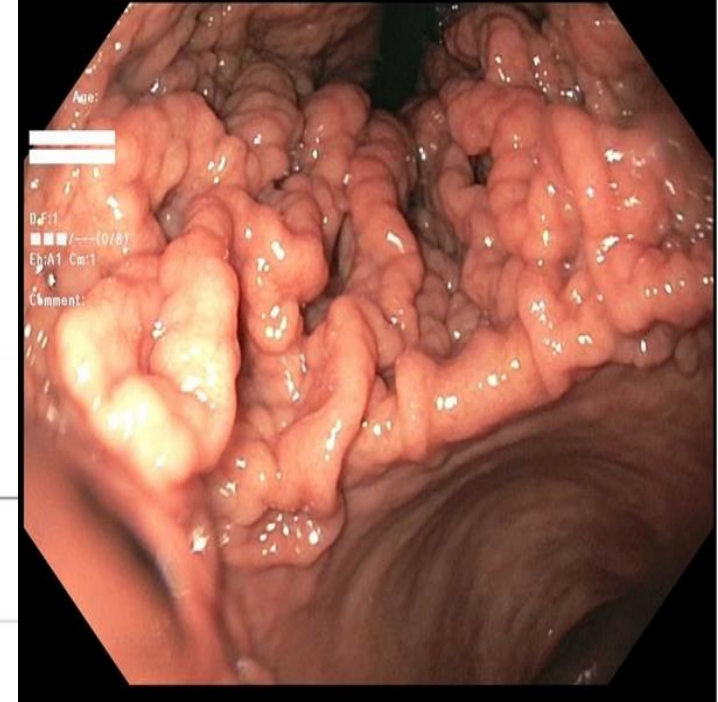
- ▶ Smoking
- ▶ Alcohol is **NOT** a risk factor
- ▶ Hyperplastic polyps : less risk factor than in colon ca (villous, >1 cm)
- ▶ Chronic atrophic gastritis (*H.pylori*)
- ▶ Chronic non- healing gastric ulcers
- ▶ Pernicious anemia
- ▶ Blood group A



# Precancerous changes

- ▶ precancerous diseases
  - ▶ chronic atrophic gastritis
  - ▶ gastric ulcer
  - ▶ gastric polyps
  - ▶ gastric remnant
  - ▶ Ménétrier's disease (hypoproteinemic hypertrophic gastropathy)
- ▶ precancerous lesion
  - ▶ atypical hyperplasia

1900 Cases		
Precancerous lesion	Number of cases	%
Hyperplastic polyp	10	0.53
Adenoma	47	2.47
Chronic ulcer	13	0.68
Atrophic gastritis	1802	94.84
Verrucous gastritis	26	1.37
Stomach remnant	2	0.11
Aberrant pancreas	0	0
Total 1900		100





# Risk factors :

## ► Genetics :

- 3% of gastric CA are inherited, most are sporadic.
- P53 (Li-fraumini).
- HNPCC (Lynch), MUTYH polyposis
- Familial adenomatous polyposis FAP./ APC mutation
- P-TEN (Cowden) hamartomas.
- BRCA-2
- Peutz-Jegher Syndrome.
- CDH-1 mutation : AD , 70% risk for hereditary diffuse gastric CA
  - need prophylactic total gastrectomy at age of 20 or 10 years before the age of the youngest diagnosed family member
  - endoscopic screening is unreliable (usually submucosal lesions)




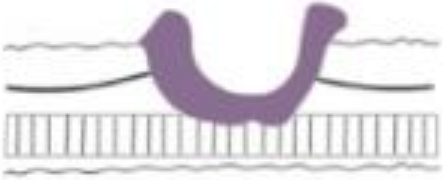


# Pathology :

- ▶ Arises from the mucus producing cells, not the acid producing ones.
- ▶ Various classifications schemes for gastric CA.
- ▶ 1- Anatomical : (according to the site):
  - Proximal (cardia) → might be treated as pure gastric or considered distal esophageal (Siewert classification)
  - Distal (non cardia) - more with H.pylori infection.
  - Diffuse (9%) , linitis plastica / leather-bottle stomach



## 2- Bormann's Classification

- Classification based on gross appearance of the lesion

POLYPOID	FUNGATING	ULCERATED	FLAT
			
<p>Systemic symptoms</p> <p>Intestinal-type</p> <p>Low grade</p> <p>Expansive growth pattern</p> <p>Less signet-ring cells</p> <p>Larger LN metastases</p> <p>HERCEPTEST 3+</p>	<p>Older patients</p> <p>Systemic symptoms</p> <p>Intestinal-type</p> <p>Low grade</p> <p>Infiltrative growth pattern</p> <p>Tumor necrosis</p> <p>Less signet-ring cells</p>	<p>Smaller size</p> <p>Mainly intestinal-type</p> <p>High grade</p> <p>Infiltrative growth pattern</p> <p>More frequent signet-ring cells</p>	<p>Younger patients</p> <p>Less systemic symptoms</p> <p>Diffuse-type</p> <p>Signet-ring cells</p> <p>Infiltrative growth pattern</p> <p>High grade</p> <p>No tumor necrosis</p> <p>No MSI</p>
		<i>Female patients</i>	<i>Female patients</i> <i>Perineural infiltration</i>



► **3- Histopathological (Lauren's classification) → the most adapted classification**

- **Intestinal type :**

- more in elderly, males.
- usually well to moderately differentiated.
- associated with metaplasia / chronic / atrophic gastritis. (H.Pylori)
- More distally localized than proximal in the stomach
- more tendency to spread through lymphatics and hematogenously.
- liver is the M.C site of mets.
- Better prognosis

- **Diffuse type :**

- more in younger , females
- usually poorly differentiated + signet ring cells
- without gastritis
- More proximal
- spread transmurally with local invasion, lymphatics and peritoneal mets.
- Usually worse prognoses



# Spread Patterns

- ▶ Direct invasion / transmural
- ▶ Lymph node dissemination
- ▶ hematogenous spread
- ▶ Transperitoneal/transcoeleomic spread



# Clinical Presentation of gastric adenocarcinoma :

- ▶ Asymptomatic, discovered incidentally
- ▶ Early gastric CA :
  - No obvious signs and symptoms.
  - Vague epigastric abdominal pain for months , Mistreated as dyspepsia or PUD.
- ▶ Progress to more obvious epigastric pain, anorexia, weight loss and vomiting +/- hematemesis (most common Sx's)
- ▶ Dysphagia in proximal tumors.
- ▶ Symptoms of G.O.O in distal tumors (indicate locally advanced disease)
- ▶ Palpable epigastric mass in 30%.
- ▶ Chronic anemia
- ▶ Sx's & Sx's of large bowel obstruction. (invasion to transverse colon)



- ▶ Signs and symptoms of metastatic disease : (40% have stage IV disease on presentation)
  - palpable left supraclavicular (Virchows) LN.
  - palpable periumbilical (Sister Mary Joseph's) nodule.
  - Blumer's shelf on DRE. (palpable peritoneal deposit)
  - ascites.
  - Jaundice.
  - Palpable ovarian mass (Krukenberg tumor) - via peritoneal seeding.
- ▶ Elective vs. Emergency presentation ( G.O.O , perforating malignant gastric ulcer, upper GI bleeding)



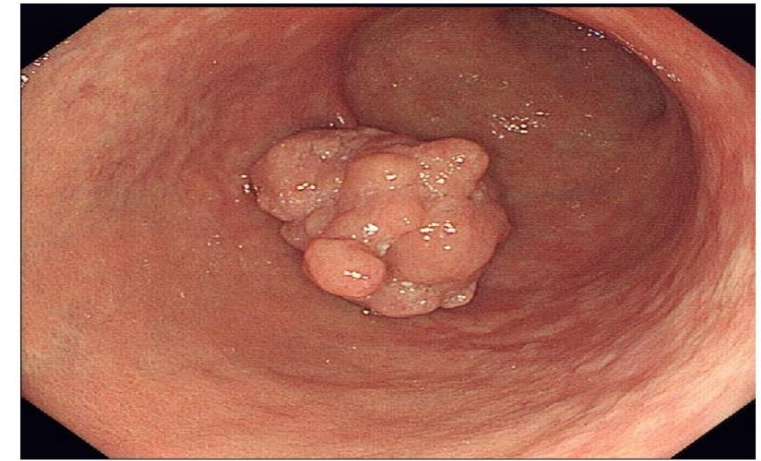
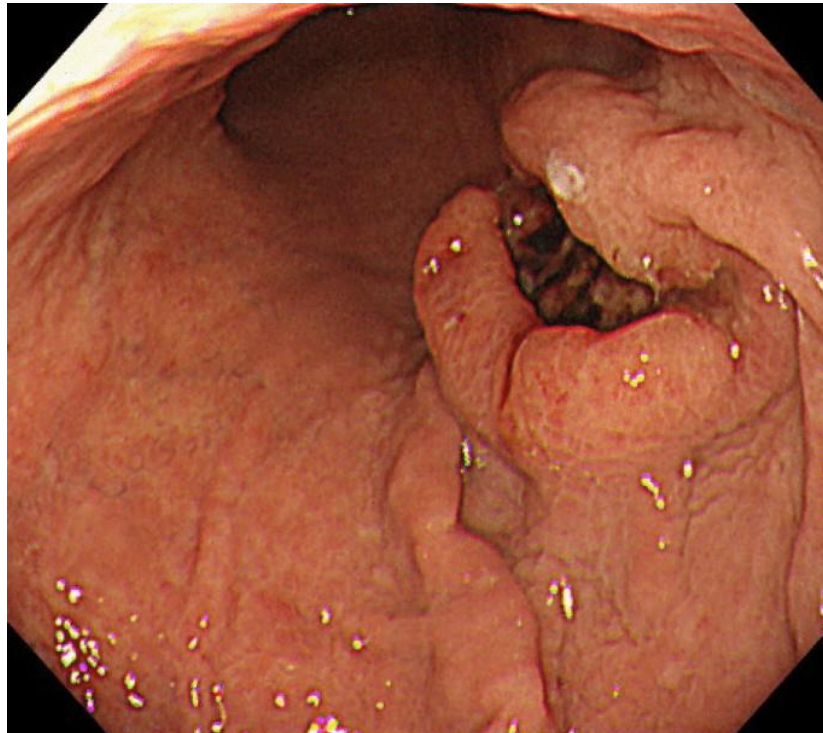
# Diagnosis / Assessment / Staging

- ▶ Full Hx and PEx (age, chronicity of pain and non response to usual antacids)
- ▶ CBC (anemia) , KFT , LFT (usually normal but can be elevated in liver mets)
- ▶ CEA :
  - High in only 30%.
  - Not useful for diagnosis / screening
  - Useful in monitoring response to treatment, surveillance for recurrence.
- ▶ Assessment of nutritional status (weight, BMI, serum albumin, pre-albumin, zinc, Mg<sup>++</sup> )
- ▶ AXR : (distended stomach in GOO, perforation, obstruction)



# ► Upper Endoscopy :

- The Gold standard for diagnosis
- Allows direct anatomical localization for planning surgery.
- Confirms Dx and histopathological features with biopsy.
- Possible stenting for obstructed inoperable disease.
- 95 % accuracy

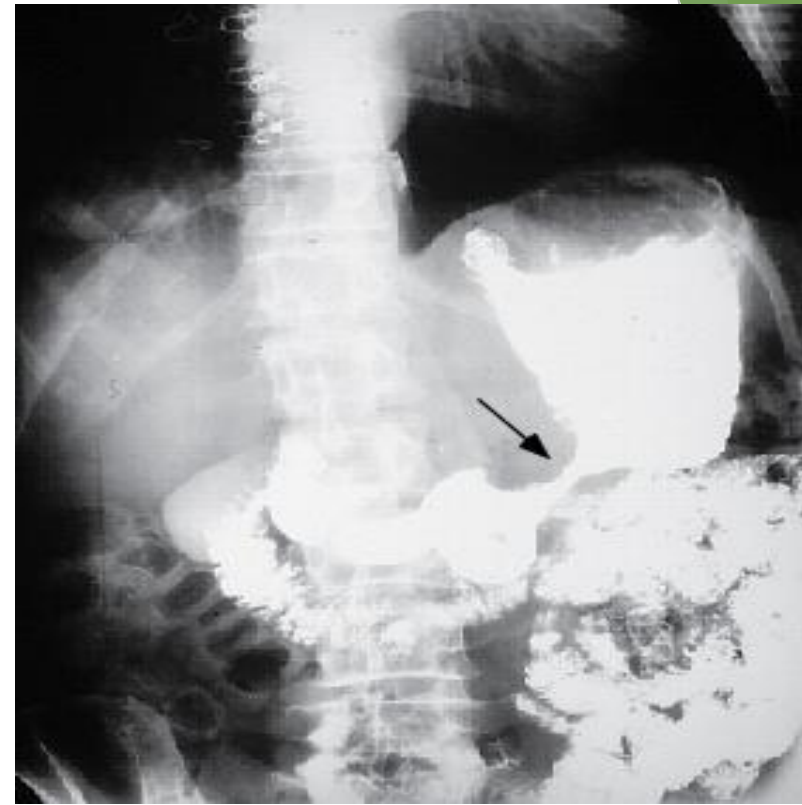
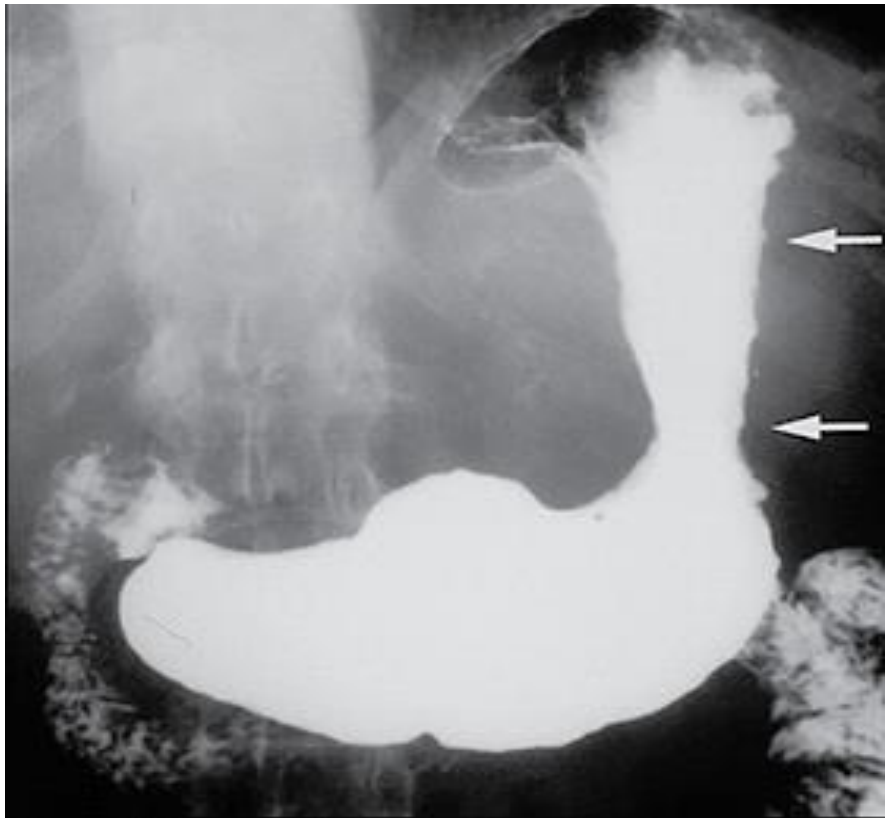




# Barium swallow

75% accuracy

for obstructive lesions only



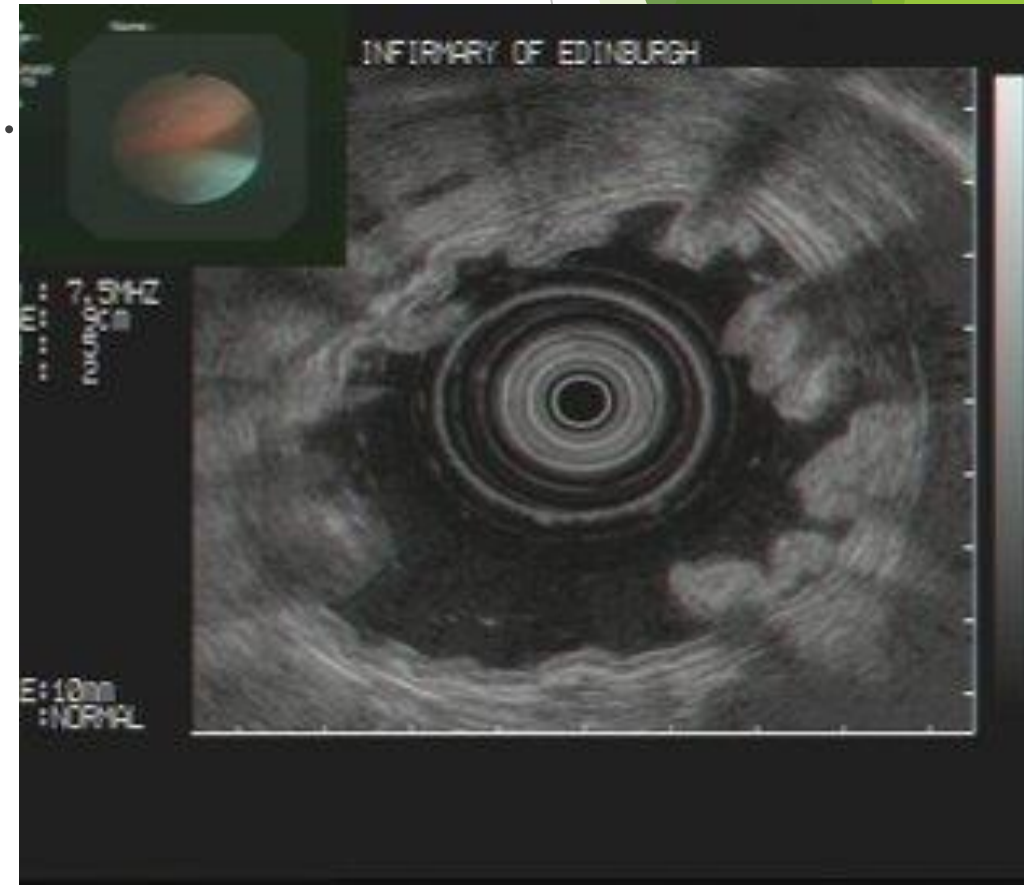
*Apple core sign"*

*Linitis plastica "leather-flask" appearing stomach*



## ► Endoscopic Ultrasound (EUS) :

- Utilizes both endoscopy + direct Ultrasound imaging.
- The most specific and sensitive for determining the 'T' stage of the tumor
- Very accurate in evaluating the local LN status 'N' stage.
- Allows sampling of local LN
- Indicated for locally advanced gastric CA.



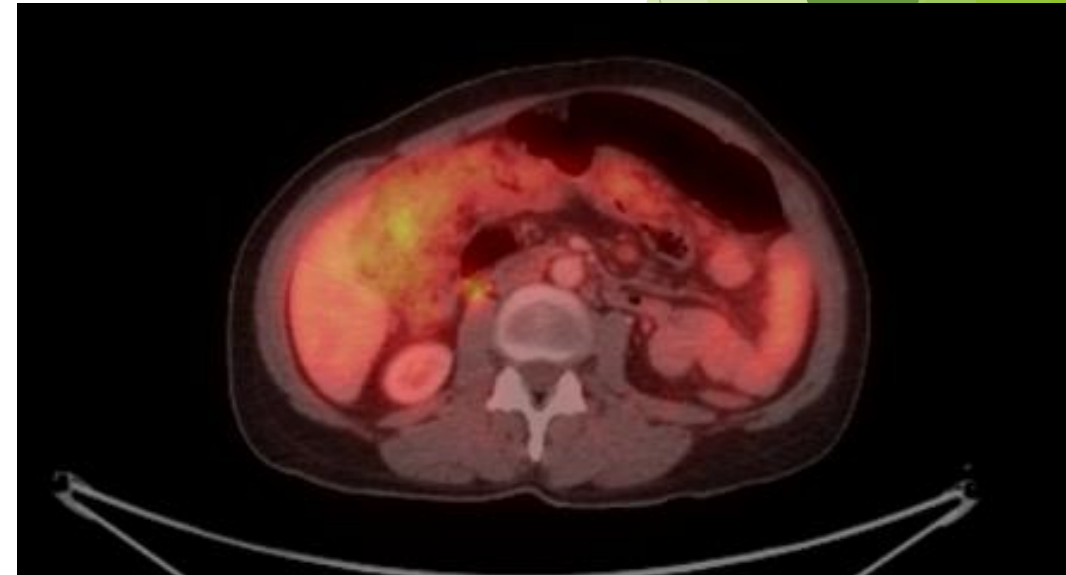


## ► Contrast CT (chest/abd/pelvis) :

- Usually for staging purposes
  - Solid organ mets, non regional LN mets
  - Ascites
  - Large peritoneal deposits
- Limitations : early gastric ca, small <5mm peritoneal and liver mets

## ► PET/CT :

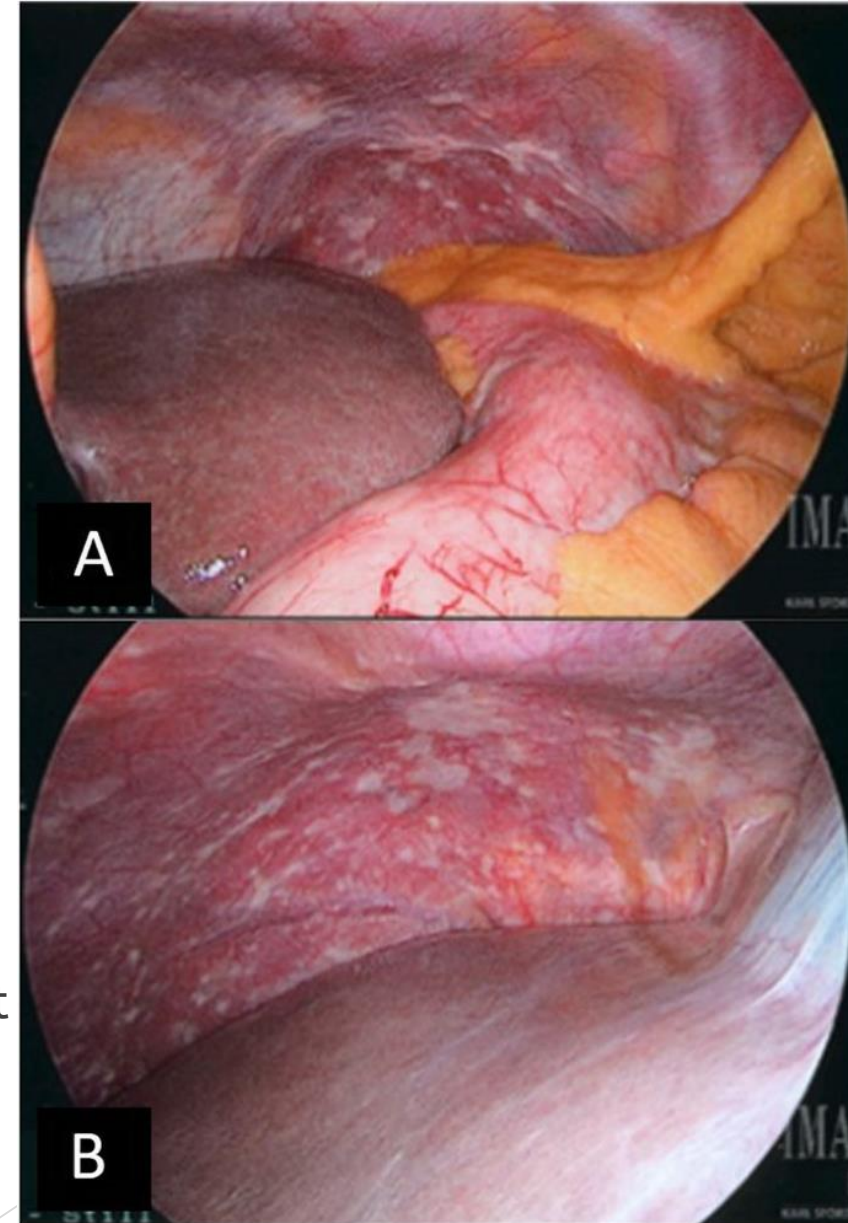
- CT combined with FDG radiolabeled glucose
- More uptake by metabolically active cells (including malignant cells)
- Improve detection of occult mets than regular CT , but still limited.





## ► Staging Laparoscopy :

- Now considered a Standard of care in locally advanced gastric CA
- Before initiation of treatment.
- Performed in O.R by inserting 1,2 or 3 trocars
- Complete inspection of the abdominal cavity for small mets (peritoneal surfaces, mesentry, under surface of diaphragm and liver surface)
- Advantages :
  - Identify small mets that are missed by CT (<5mm).
  - Obtain tissue biopsy of suspicious nodules
  - Perform peritoneal lavage and fluid cytology
  - Local disease status (involvement of adjacent organs)
- Among patients deemed to have non metastatic disease initially, 36% of patients were actually stage IV disease which alter the treatment (occult peritoneal mets 21%, +ve wash cytology 13%, liver mets 6%) - in a study performed at *MD Anderson cancer center -Texas, USA*.
- Might need to be repeated after NACTX, before definitive surgery.





# Staging of Gastric Adenocarcinoma (TNM)

► Necessary for 1-determining extent of disease, 2-planning of tx, 3- reflecting prognosis.

► (T) :

- Tis : carcinoma in situ, high grade dysplasia/ confined to mucosa
- T1 : tumor invading submucosa/ **lamina propria**.
- T2 : tumor invading **muscularis** propria.
- T3 : tumor invading subserosa without visceral peritoneum.
- T4 : tumor invading serosa (visceral peritoneum) or adjacent organs.

► (N) :

- N0 : no evidence of local LN involvement, N1 : 1-2 LN, N2: 3-6 LNs, N3 : 7 or more LNs.
- +ve LN makes it stage III

► (M): → M0 : no distant mets, M1: distant mets (stage IV)

► Mets to non-regional LNs (mesenteric, para-aortic, portal, retroperitoneal) is M1 (stage IV)

► +ve abd wash cytology is M1 (stage IV)

► Staging might change after surgery



# Treatment :

- ▶ Various factors contribute to treatment selection:
  - Clinical stage of the disease
  - Patient related factors (Age, co-morbidities)
- ▶ Possible treatment options :
  - Chemotherapy (Neoadjuvant, Adjuvant, palliative)
  - Radiotherapy (only for palliation)
  - Endoscopic resection (very early disease, selected patients)
  - Surgical resection (mainstay of treatment, the only curative one)



# Endoscopic Mucosal Resection (EMR)

- ▶ Only for early disease confined to mucosa (Tis, T1a)N0M0
- ▶ Low probability of LN mets (0-4%)
- ▶ Absolute indications : well differentiated, <2cm, no ulceration, T1a
- ▶ Offers survival similar to surgical resection if performed for absolute indications.
- ▶ 5 yr- survival 95%
- ▶ Slightly higher incidence of local recurrence (6% vs 1% for surgical resection)
  
- ▶ Advantages : less invasive, less morbidity , optimal for old frail patients with co-morbidities.
  
- ▶ Limitations :
  - performed only in specialized high volume centers with good experience (Japan)
  - Need closer surveillance / followup

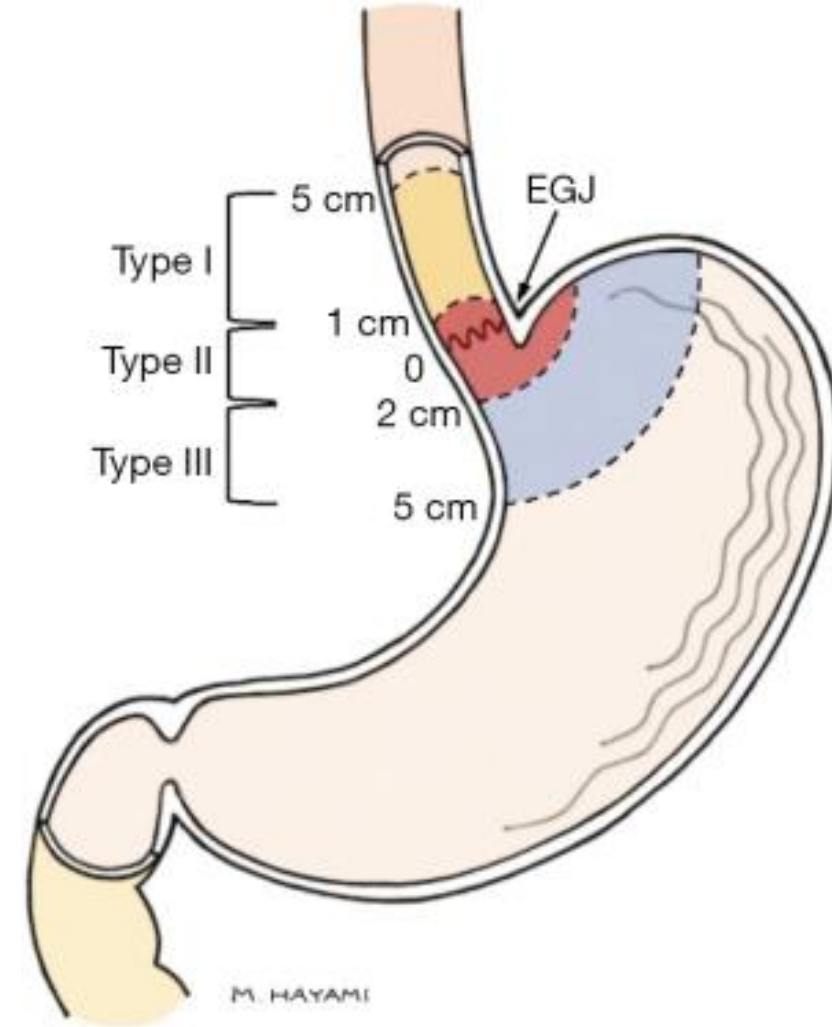


# Surgical Treatment :

- ▶ The mainstay of treatment and the only curative modality (along with EMR).
- ▶ **GOAL** is to remove the tumor with at least 5cm tumor free margins and performing routine Lymphadenectomy.
- ▶ Extent of gastrectomy depends on the anatomical location of the tumor and might include **total/ subtotal or distal gastrectomy**
- ▶ for **distal** (body and antrum) tumors , distal or subtotal gastrectomy leaving a remnant proximal gastric pouch can be feasible as long as the 5 cm -ve margin is obtainable.
- ▶ Otherwise , total gastrectomy.
- ▶ All have similar outcome and survival.
- ▶ Partial gastrectomy has a better quality of life and post op nutritional status.



- ▶ Proximal tumors : (cardia and GEJ)
    - If <2cm below GEJ → treated as esophageal not gastric
  - Siewert classification : type1 (1-5cm above GEJ)  
Type2: (2cm below) are esophageal
  - Type 3 (2-5 cm into the stomach are gastric CA)
- ▶ All proximal pure gastric CA should undergo total gastrectomy (not partial)

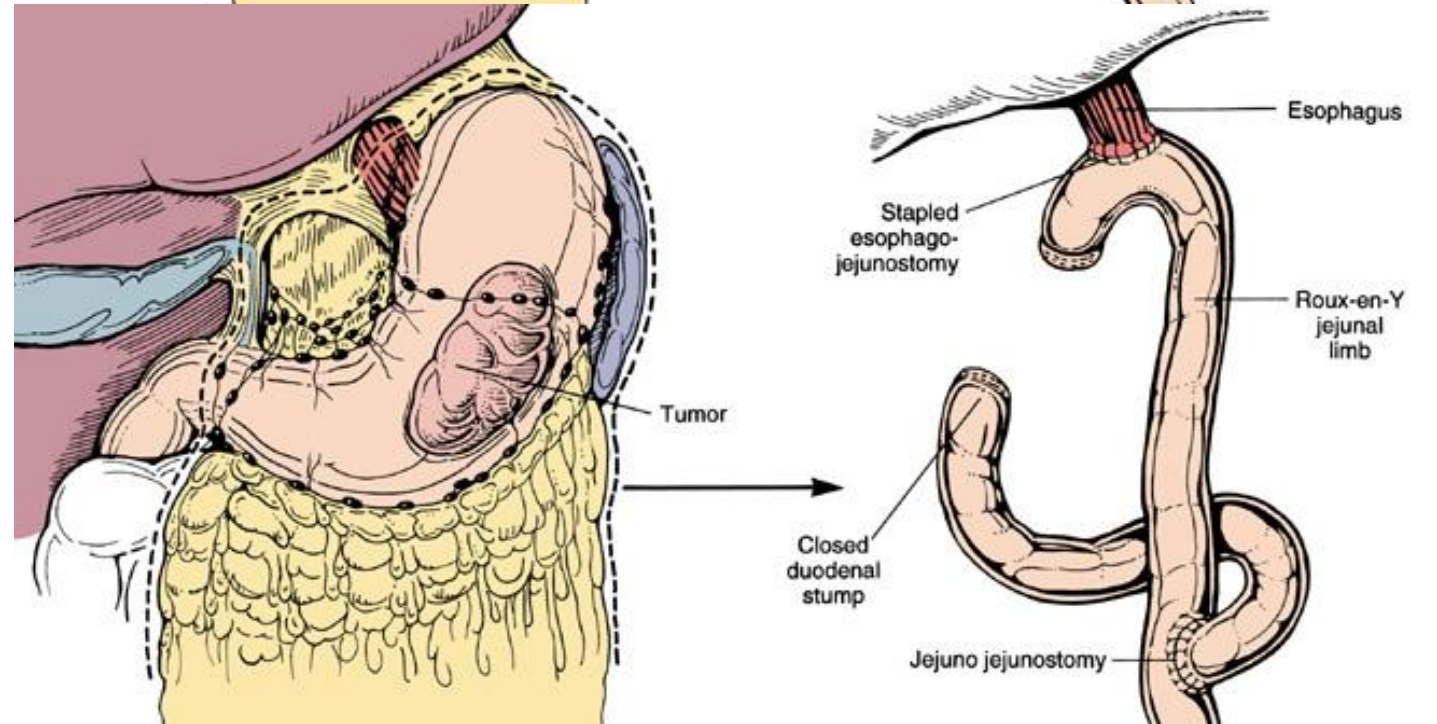
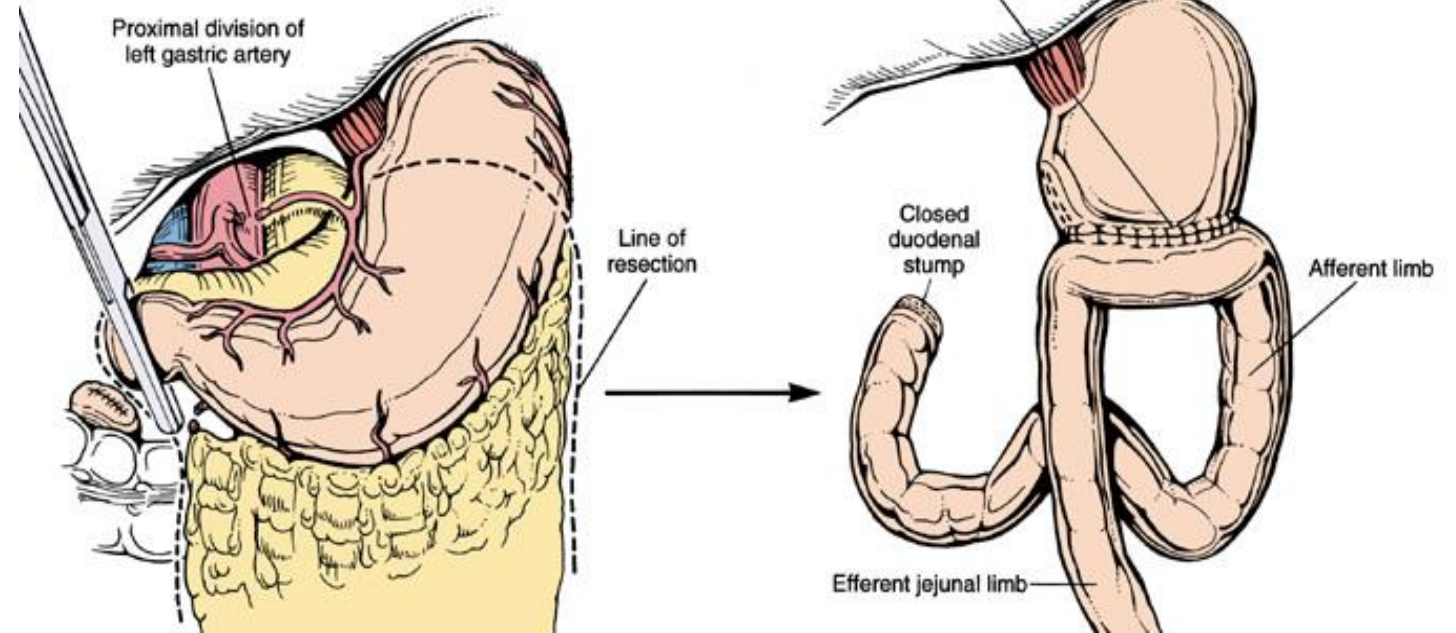
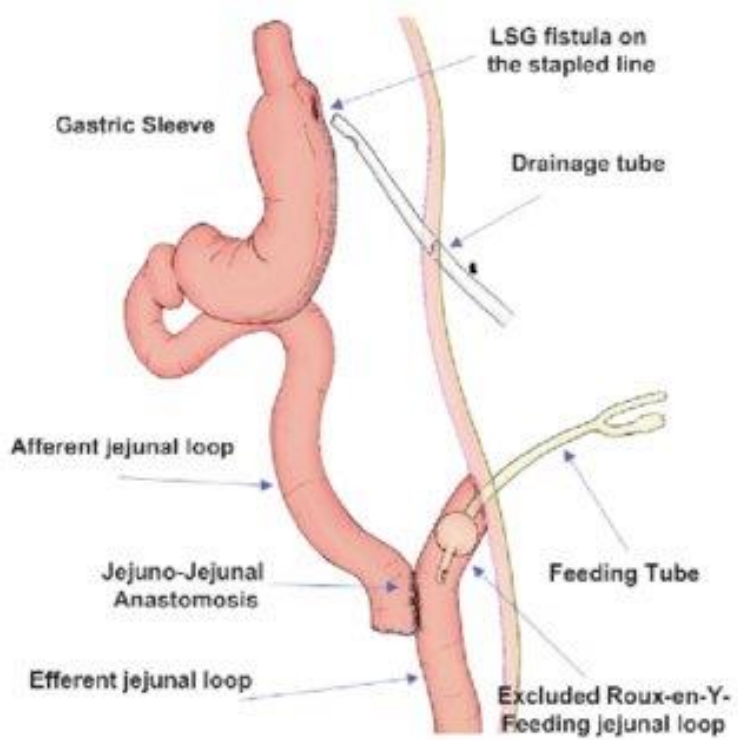




- ▶ Total gastrectomy : (for proximal and midbody tumors)
  - Removal of the whole stomach and reconstruction with **ROUX en-Y** configuration
  -
- ▶ Partial (distal/subtotal) gastrectomy: (for antral/distal tumors)
  - Removing part of the stomach with remaining proximal gastric pouch (supplied by short gastric vessels).

reconstruction can be done with **Billroth II** OR with **ROUX en-Y** configuration.
- ▶ **open vs. laparoscopic**
- ▶ Both offer same survival/outcome/completeness of resection.
- ▶ Lap. Surgery has lower post op pain, faster recovery and earlier return to daily activities. But require a more skilled surgeon.
- ▶ Feeding jejunostomy insertion at surgery for all patients.

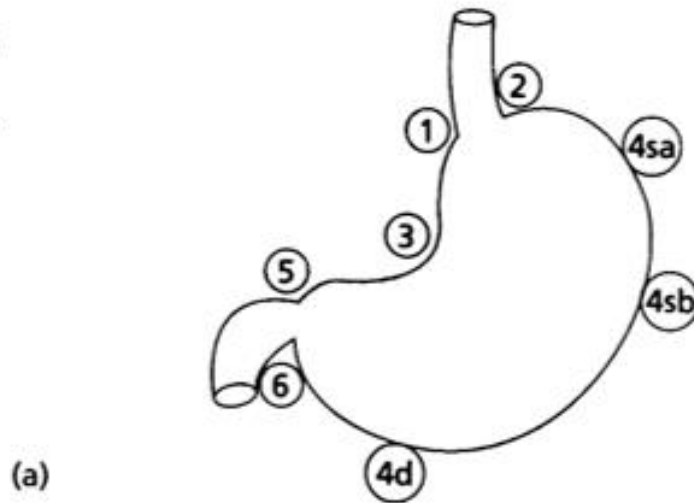






- ▶ **Lymphadenectomy (LN dissection) :**
- ▶ At least 16 LN , for accurate staging and cure.
- ▶ The standard is to remove level **N1** (perigastric LNs) and **N2** (celiac, C Hepatic, left gastric, splenic LNs) → **D2 lymphadenectomy.**
- ▶ D zero is incomplete lymphadenectomy,
- ▶ D1 is removal of N1 perigastric LN (all nodal tissue within 3 cm of the primary tumor (might be sufficient in early CA)
- ▶ **D2** is the standard in gastrectomy for cancer
- ▶ D3 (D2++ paraortic,retropancreatic) → higher morbidity, no added survival benefits
- ▶ D3 only performed in Japan
  
- ▶ Japanese literature shows survival benefit of extended D2 dissection:
- ▶ 39% 5 year survival after D2 vs. 18% for D1
  
- ▶ Western literature shows no survival benefit but increased morbidity and mortality of D3 dissection

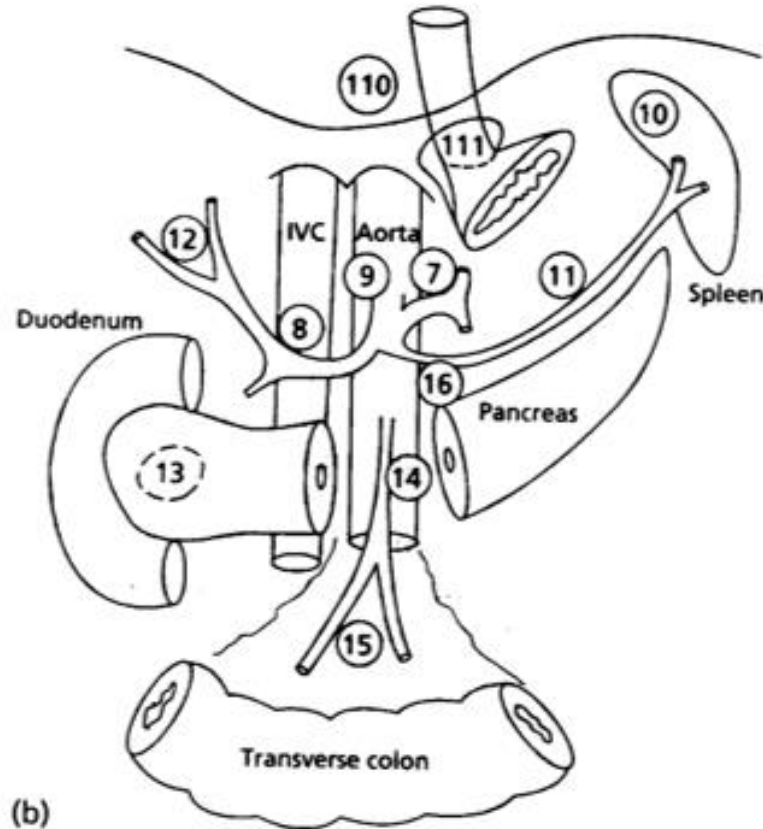




(a)

### Key

- 1 = right cardiac
- 2 = left cardiac
- 3 = lesser curvature
- 4 = greater curvature :
  - sa = short gastric
  - sb = left gastroepiploic
  - d = right gastroepiploic
- 5 = suprapyloric
- 6 = subpyloric



(b)

### Key:

- 7 = left gastric artery
- 8 = common hepatic artery
- 9 = coeliac axis
- 10 = splenic hilum
- 11 = splenic artery
- 12 = hepatoduodenal artery
- 13 = retropancreatic
- 14 = root of mesentery
- 15 = middle colic artery
- 16 = para-aortic
- 110 = paraoesophageal
- 111 = diaphragm



► **Splenectomy :**

- Only if spleen or splenic hilum are involved , no rule for prophylactic routine splenectomy

► **Multivisceral organ resection : (e.g. pancreas, transverse colon)**

- For T4 tumor involving adjacent organ/s
- Only if you can get -ve margins, patient can tolerate it, and no mets



# Post op. complications

## ► General complications : 20-40%

- Pulmonary (atelectasis, pneumonia).
- Bleeding (intra op or post op)
- Infection (SSI)
- Cardiac (MI, arrhythmias)
- Venous thromboembolism (DVT, PE)

## ► Specific to gastrectomy:

- Anastomotic site leak
- Post op ileus
- Dumping Syndrome
- Nutritional impairment (B12, calcium, iron deficiency, Food restriction)
- Diarrhea
- Gallstones
- Stomal ulceration, anastomotic site stricture



# Anastomotic site leak

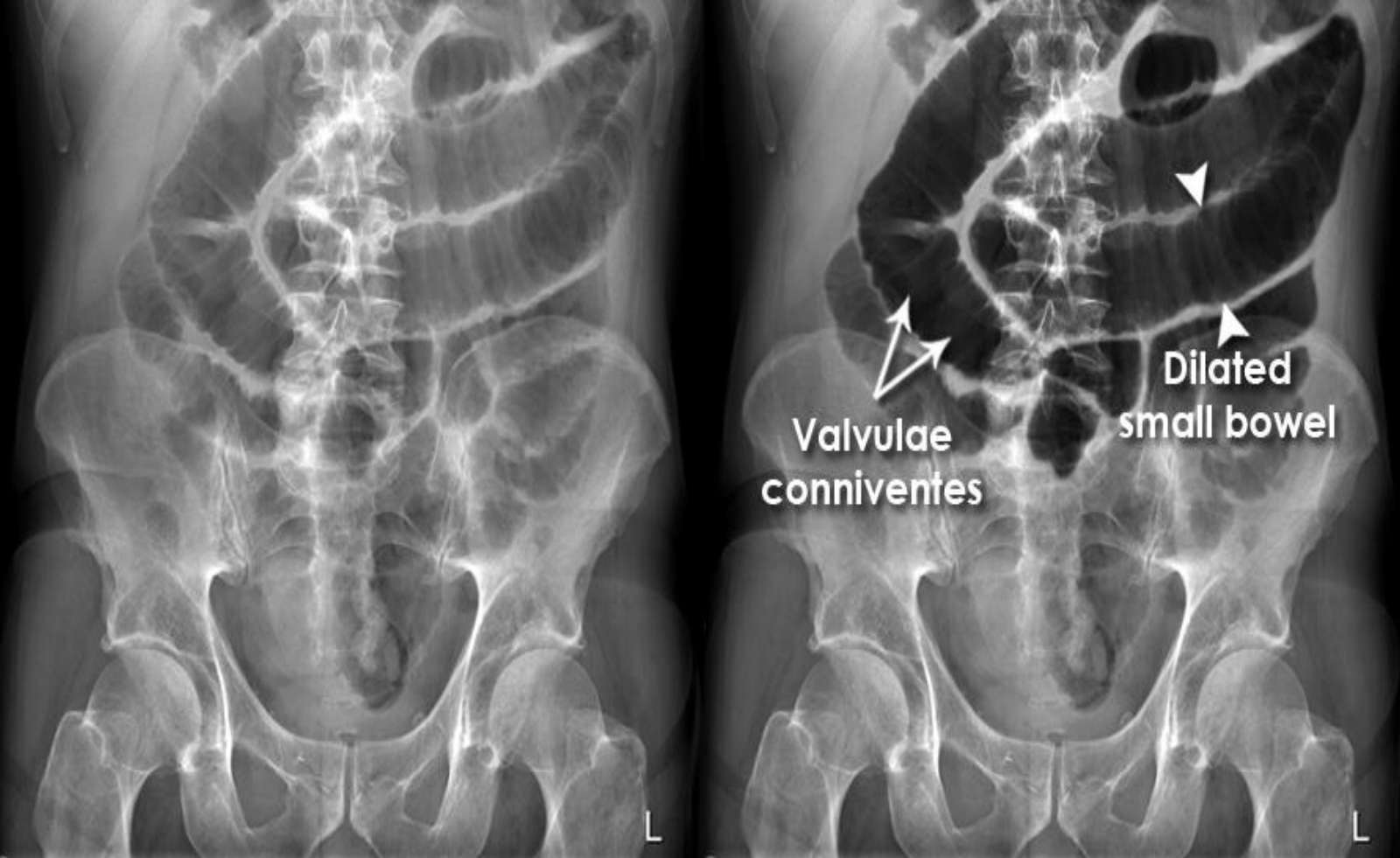
- ▶ 2-10%
- ▶ Can be early or late (up to 40 days post op)
- ▶ Leak in the first few days is usually a technical failure
- ▶ Dx : fever, leukocytosis, tachycardia, signs of peritonitis or food/juice in drains if free leak
- ▶ CT with oral contrast or GG swallow detects leak
- ▶ Some surgeons perform routine contrast imaging before initiation of oral feeding (post op day 5)
- ▶ Management :
  - non-surgical (antibiotics, nutritional support, endoscopic covered stents or percutaneous CT guided drainage (if contained leak)
  - Surgical : re-laparotomy and revision of anastomoses .



# post op ileus :

- ▶ More with open surgery
- ▶ Due to extensive bowel manipulation during surgery
- ▶ Electrolytes imbalance narcotic analgesics and leak can be a cause too.
- ▶ bowel motion should return to normal 4-6 days after surgery
- ▶ Sx&Sx's : recurrent bilious vomiting, abd distention, failure to pass gas or stool, sluggish bowel sounds
- ▶ AXR shows diffusely dilated small bowel loops and multiple air-fluid levels
- ▶ Treatment : Keep NPO, treat the cause, early ambulation of patient.







# Feeding post op :

- ▶ By jejunostomy feeding tube connected to a pump providing a liquid formula (ENSURE) starting from day 2-3
- ▶ Start oral feeding on day 4-7 (gradually from fluid to solid )
- ▶ Some surgeons perform oral contrasted studies routinely before oral feeding.
- ▶ May take months to return to near- normal feeding habits.
- ▶ Rule out leak and ileus before.



# Chemotherapy for treatment of gastric adenocarcinoma :

## ► Neoadjuvant chemotherapy : (NACTx)

- Multiple different regimens , FLOT is the most recent one
- for any patients with locally advanced non metastatic disease on clinical pre-op staging T3/T4, N+, M0 (stage II, III)

## ► Benefits from NACTx :

- Improve resectability and chance of getting a negative margin resection.
- Test tumor behaviour/biology (aggressive vs non aggressive)
- Decrease recurrence and improve survival in locally advanced disease.
- Avoid delay in treatment with chemo that might occur after surgery if developed complications post op.

## ► Adjuvant chemotherapy :

- Given as a continuation of pre op chemo regimen or for those who have higher stage than before surgery (pts who were thought to be less than T1/2,N-)



# Metastatic gastric adenocarcinoma

- ▶ 40% of pts present with metastatic disease at diagnoses.
- ▶ 36% of patients who were initially deemed to have a non metastatic disease found to be metastatic
- ▶ Some patients might be non metastatic but not fit for curative treatment and surgery (severe comorbidities, locally unresectable disease)
- ▶ Some pts may have recurrence after curative treatment
- ▶ The goal of treatment in all of these patients is only palliative (improve symptoms and quality of life only)- less chance of bleeding, perforation, relief of obstruction, pain and improve nutrition



## ► Palliative treatment options :

- Chemotherapy (minimal prolongation of survival, improve symptoms)
- Radiotherapy (decrease tumor size, less chance of bleeding/obstruction/perforation)
- Ascites : repeated tapping.
- Endoscopic : (stenting for obstruction, management of bleeding)
- Surgical :
  - Palliative bypass (gastrojejunostomy)
  - Gastrostomy tube for decompression
  - Palliative resection not advised for most of patients



## ► Survival :

- Depends on stage, completeness of resection (if candidate)
- 5 yrs- survival :
- local disease 50-80%,
- +ve LN 30%,
- Metastatic 5%



## ► Recurrence :

- High even after complete resection (30%)
- Most occur in the first 2 years (80% of recurrences)
- Can be local (surgery site) ,
- distant (liver) ,
- peritoneal mets
- Carry a dismal prognoses (6 months survival)
- Treated as palliative only (usually chemotherapy) or other modalities of palliative treatment



## ► Post op follow up / surveillance:

- In clinic every 3 months in the first 2 years , then every 6 months
- Include full Hx, PEx, labs
- Endoscopy at 1,2,5 years if partial gastrectomy only
- CT scan every 6 months in the first 2 years , then annually



# Gastric Lymphoma :

- ▶ 5% of gastric neoplasms.
- ▶ The Stomach is the commonest extranodal primary site for non-Hodgkin's lymphoma
- ▶ Secondary Lymphoma is commonly seen in stomach from another site
- ▶ The histologic types
  - low-grade B-cell lymphomas of the mucosa-associated lymphoid tissue (**MALT**) type.
  - high-grade diffuse large B-cell lymphoma (**DLBCL**);
  - **Mixed** containing both
- ▶ chronic gastritis and infection with H. pylori is the main predisposing factor
- ▶ Symptoms :
  - ▶ pain (88%), anorexia(47%), weight loss (25%), bleeding (19%), and vomiting (18%).
  - ▶ Obstruction and perforation, Anaemia and epigastric mass.



# Investigations

- Labs: CBC, LDH , B2-macroglobulin, bone marraow biopsy
- Staging CT
- Endoscopy + biopsy (histology + test for H.pylori)
- Might need deep biopsies
- EUS is the best modality.



# Treatment :

- ▶ Early low grade MALT lymphoma → *H.pylori* eradication therapy
- ▶ Assessment of response with endoscopy, most pt will have complete remission
- ▶ persistent MALT lymphoma after *H. pylori* therapy → external beam radiation (90% to 100% rate of complete response)
- ▶ Single agent chemotherapy +/- Rituximab
- ▶ For DLBCL : combination of chemotherapy (R-CHOP)
- ▶ Autologous stem cell transplantation
- ▶ surgical intervention in bleeding, perforation, obstruction, and residual disease after primary therapy.
- ▶ 5 yrs-survival : 90% for low grade, 50% for high grade tumors.



# Gastrointestinal Stromal Tumors (GIST)

- <1 % of gastric CA
- mesenchymal tumors
- Arises from interstitial cells of Cajal (pacemakers of GI motility)
- 95% stain with Ab against c-KIT (CD117) 70% stain for CD34
- 3-5% of the remainder of KIT-negative GISTs contain PDGFR-alpha mutations.
- Solitary , grows extraluminally to a large size
- Variable clinical behavior, from slow-growing indolent tumors to aggressive malignant cancers with the propensity to invade adjacent organs, metastasize to the liver, and recur locally within the abdomen.
- Site, size (>5cm) and mitoses(>5HPF) determines indolent vs more aggressive tumors
- The stomach is the most common site of GIST. Other includes small bowel, colon and rectum



# GIST tumors

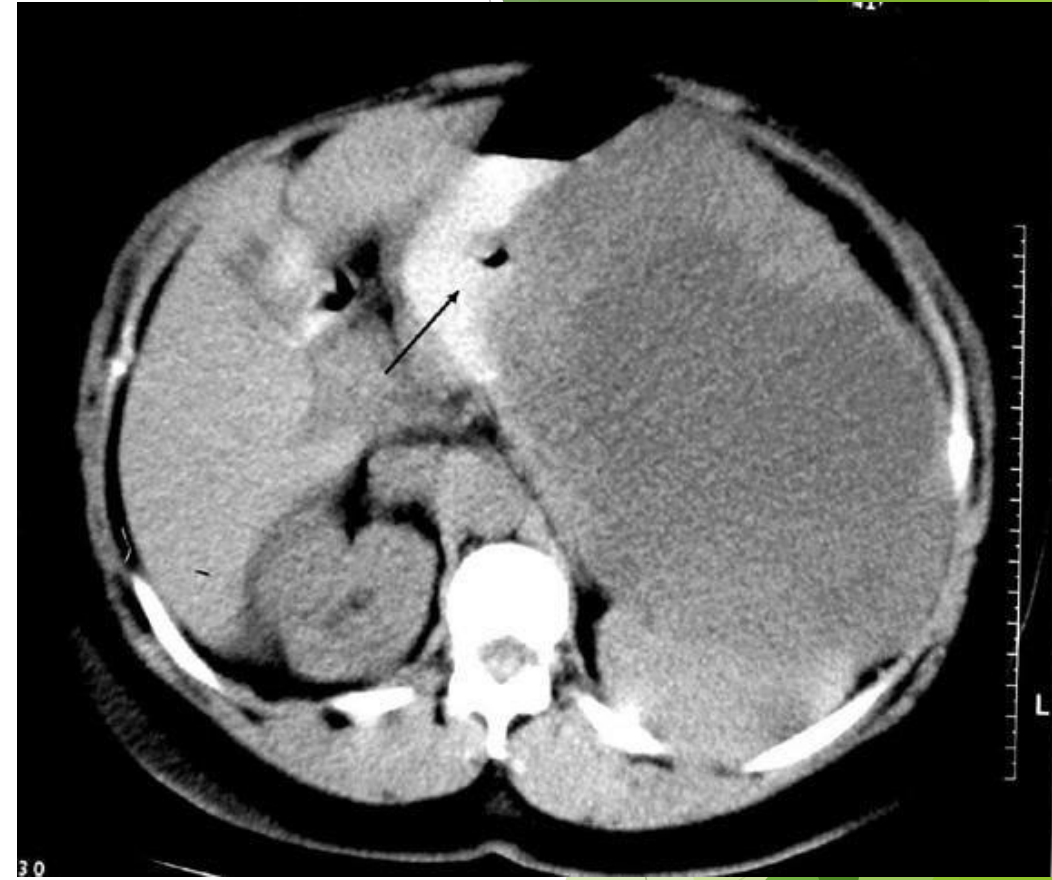
## ► Presentation:

- might be asymptomatic/ discovered incidentally or
  - Abdominal pain
  - Anorexia
  - Nausea, Vomiting
  - Weight loss
  - Epigastric fullness
  - Early satiety
  - Bleeding/perforation/obstruction



# Investigations:

- ▶ Routine Labs
- ▶ Barium swallow, U/S , and AXR gives limited information
- ▶ CT scan → very important to obtain
  - site, size, characteristics of tumor, relation to surrounding organs, mets)
- ▶ Endoscopy : (sometimes misses submucosal lesions)
- ▶ EUS better than endoscopy , also can obtain biopsy but limited value in large lesions.
- ▶ Biopsy is not always indicated if dx is suggestive and surgery is needed anyway





# GIST tumours

## ► Treatment

### ► Surgery-local excision of tumor with 1cm -ve margins

- Lymph node clearance unnecessary as spread is not common
- Large tumors may need formal gastrectomy+/- adjuvant therapy (imatinib)
- Tumor rupture/spillage during surgery and positive margin significantly **worsens** the prognoses

### ► Medical therapy consists of tyrosine kinase inhibitors (TKI), c-KIT inhibitor imatinib mesylate (Gleevec).

- Preoperatively, to enable resectability or decrease morbidity by shrinking the tumor
- After surgery, to decrease recurrence
- In metastatic disease, as definitive treatment
- In recurrent, unresectable disease, as definitive treatment



